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CONTENTS

		PAGE
Recession of the trochlea	*W. L. Hughes and D. W. Bogart*	911
Ocular lesions of lymphogranuloma venereum	*C. Espíldora and W. E. Coutts*	916
Epithelial tumors of the limbus	*J. E. Ash and H. C. Wilder*	926
Tetanus following ocular injury	*J. O. Wetzel*	933
Atypical and typical achromatopsia	*L. L. Sloan and S. M. Newhall*	945
Melanoma of the choroid	*G. R. Callender, H. C. Wilder, and J. E. Ash*	962
Chronic ophthalmoplegia externa	*I. D. Fagin*	968
Transposition of extraocular muscles	*P. J. Leinfelder and N. M. Black, Jr.*	974
Fascial suspension of the eye	*C. M. MacKenzie*	977
Actinomycosis of the inferior lacrimal canaliculus	*A. J. Brinckerhoff*	978
Plastic suture plates	*C. Berens*	981
Drainage for chronic tear-sac infection	*D. J. Morgenstern*	982
Staphylococcus-aureus conjunctivitis	*M. B. Lyon*	983
Bilateral exudative retinitis	*S. A. Fox and M. L. Gelfand*	984
A flexible tip for the giant magnet	*L. F. Carter*	986

DEPARTMENTS

Society Proceedings	987
Editorials	996
Book Notices	1006
Abstracts	1010
News Items	1026

For complete table of contents see advertising page V

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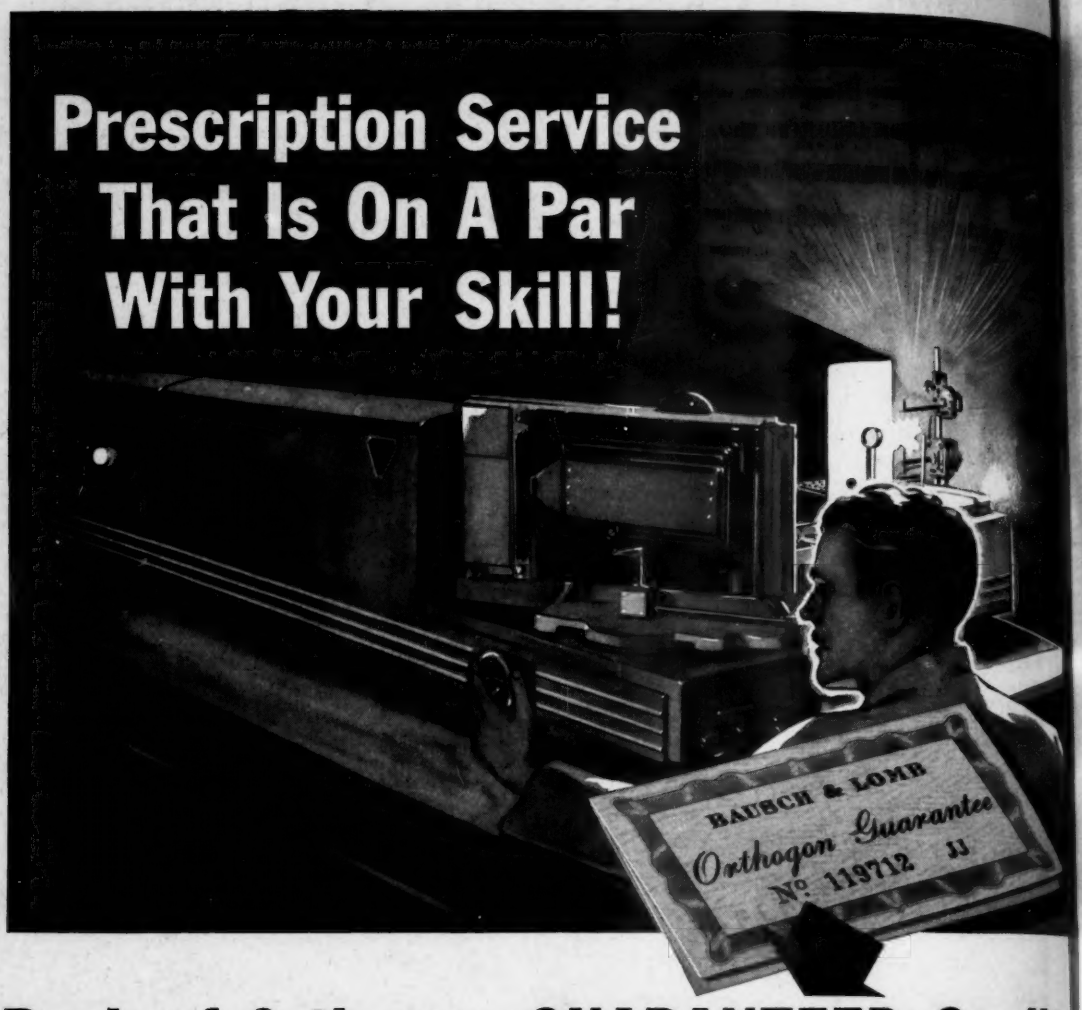

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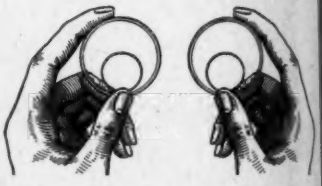

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 25 AUGUST, 1942 NUMBER 8

RECESSION OF THE TROCHLEA IN OVERACTION OF THE SUPERIOR OBLIQUE*

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Overaction of the superior oblique muscle, while not a common condition, may be the cause of extreme discomfort to the patient. Among the objective and subjective signs are head tilt, due to effort on the patient's part to line up his vertical image imbalance, and vertical diplopia, often with extorsion of one image. The diplopia is more marked in the lower nasal field of the affected eye and the torsion, of course, becomes more marked as the eye is taken into the temporal field. Effort to maintain fusion in the lower fields, especially in reading, may give rise to many and varied symptoms.

In the past, the surgical condition presented by overaction of the superior oblique muscle has been handled in several ways. The yoke muscle, the inferior rectus of the opposite eye, whether underactive or not, has been strengthened by resection, advancement, or a cinch type of operation. The opposing muscle, the inferior oblique of the same eye, has been strengthened in an effort to hold back its overacting opponent. Neither of these procedures attempted to restore the normal function of the overacting superior oblique muscle itself.

Surgery on the muscle itself has been described by the following three surgeons:

(a) Axenfeld (Arch. f. Ophth., 1921, v. 105, pp. 1197-1206):

"Cosmetic immobilization of the eye and tenotomy of the levator palpebrae superioris with comments on operations on the musculi obliqui." He reported myotomy of the inferior oblique followed by "extirpation" of the trochlea for cosmetic purpose by "exclusion" of both obliques. He stated further that to his knowledge no attack on the superior oblique muscle had ever been made. He added that retroplacement of the oblique muscle could be effected by inserting one of two longer or shorter silk loops between the ends of the muscle as in Stephenson's limited tenotomy.

(b) Van der Hoeve (Augenärztliche Operationslehre. 1922, v. 2, p. 1633):

"Tenotomy of the superior oblique is now scarcely ever performed. One way, according to Czermak and Elschnig, is to progress from the conjunctiva after detachment of the superior rectus." He also quoted Axenfeld as noted above.

(c) Wheeler (Trans. Amer. Ophth. Soc., 1934, v. 32, pp. 237-244; and Amer. Jour. Ophth., 1935, v. 18, Jan., pp. 1-5) discussed his operation for advancement of the underacting superior oblique with case reports. He severed the superior rectus, isolated the superior oblique and advanced it by placing sutures in its outer third and suturing this portion to the sclera beyond and temporalward to its insertion.

Unaware of procedures (a) and (b) for reducing the effect of the superior oblique, one of us (W. L. H.) devised a simple and quite effective method of accomplishing this objective in 1934. This

* Presented before the New York Academy of Medicine, Section of Ophthalmology, on February 16, 1942.

procedure resulted in a more normal action of the superior oblique muscle (see the muscle plottings of the cases reported). The hypotropia and intorsion of the affected eye was reduced, and in both cases the patients were relieved of their annoying symptoms.

The procedure, recession of the trochlea, is described as follows:

Anesthesia may be local or general. The former was carried out in both cases here reported by infiltration with novocaine solution to anesthetize the skin, periosteum, and the region about the

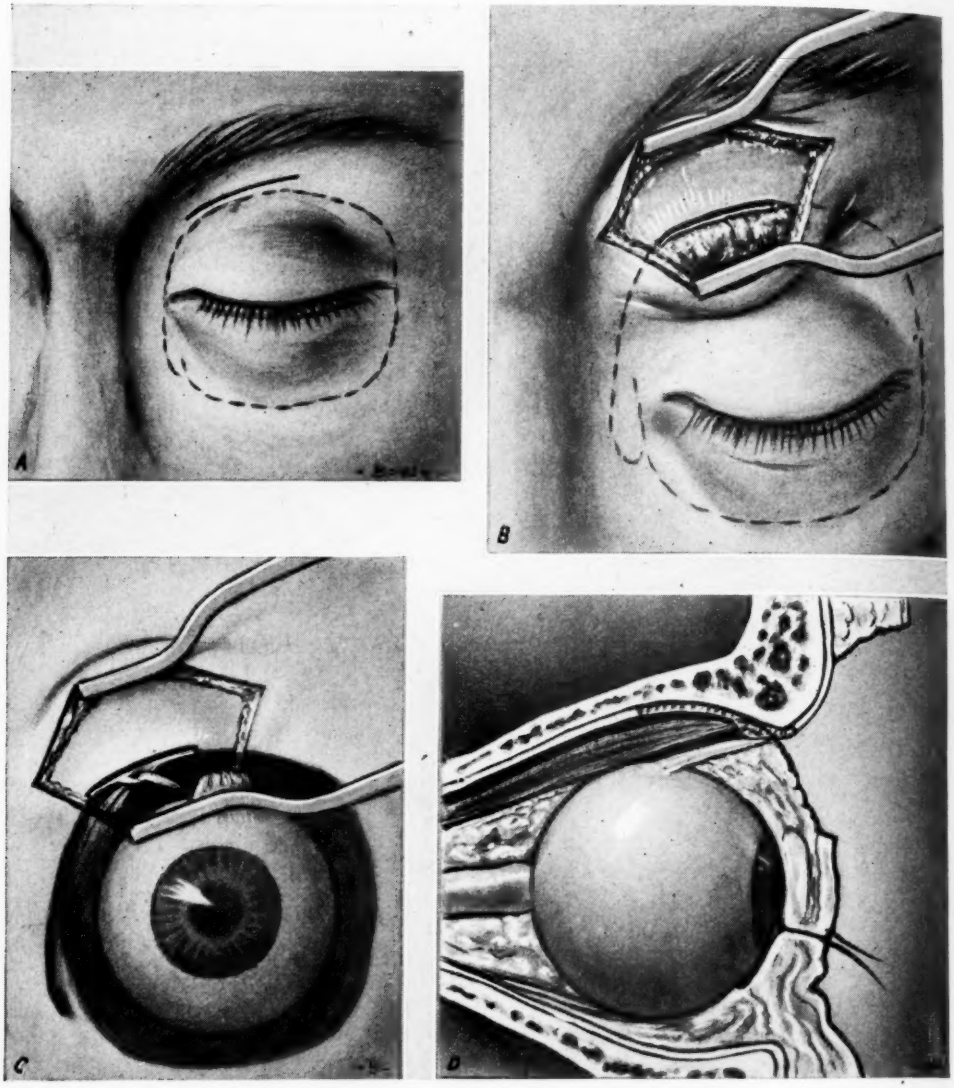

Fig. 1 (Hughes and Bogart). Recession of trochlea: A, site of skin incision in upper nasal angle over the site of the trochlea located by palpation before injection of local anesthetic. B, location of the incision in the periosteum on the margin of the orbit. C, schematic view showing the location of the trochlea in relation to the incision in the periosteum. D, cross section showing periosteum to be undermined.

trochlea. The injections were carried up to and into the periosteum on the roof of the orbit in the upper nasal angle. A transverse incision was made through the skin, parallel to and over the orbital rim (fig. 1A) in the region of the supraorbital notch. This incision over the trochlea was carried through the skin and superficial fascia, exposing the periosteum over the orbital rim. The periosteum was incised (fig. 1B) and undermined with a periosteal elevator along the roof of the orbit, 2.5 cm. back from the anterior rim (figs. 1C, D, and 2A). Parallel incisions were made on each side of the supraorbital notch about 1 cm. apart and carried deeply 1.5 cm. (These latter incisions were on each side of the trochlea, which was located by palpation previous to the injection of the local anesthetic.) The periosteum with the trochlea attached was simply pushed back (fig. 2B). The muscle and skin were closed in layers and a small plug of gauze or cotton was placed over the area of the trochlea. A pressure dressing was then applied to maintain the retroplacement of the trochlea (fig. 2C). The dressing and

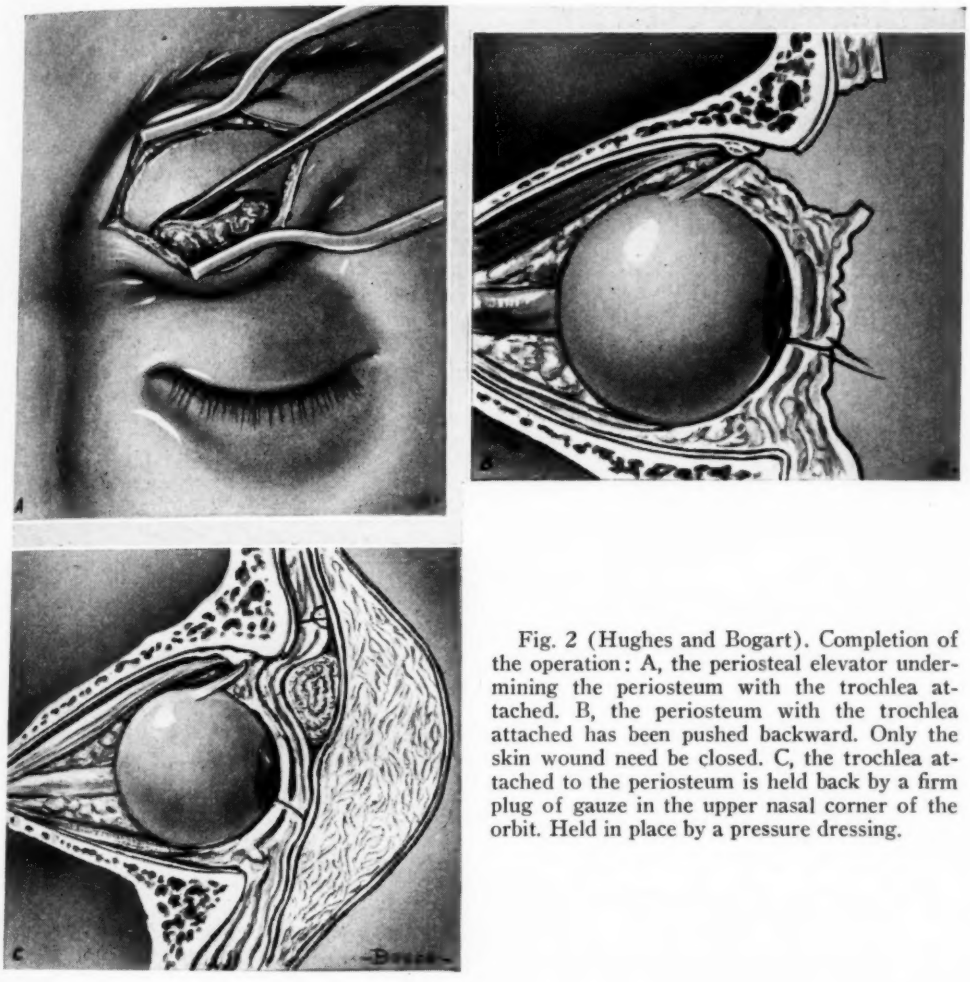

Fig. 2 (Hughes and Bogart). Completion of the operation: A, the periosteal elevator undermining the periosteum with the trochlea attached. B, the periosteum with the trochlea attached has been pushed backward. Only the skin wound need be closed. C, the trochlea attached to the periosteum is held back by a firm plug of gauze in the upper nasal corner of the orbit. Held in place by a pressure dressing.

sutures were removed in four days and a similar pressure dressing reapplied for another four or five days. The dressing was then left off.

Case reports

Case 1. Mr. R. B., aged 16 years, since childhood had tilted his head and had diplopia when looking to the left and down with the head held upright. Pictures taken when he was quite young show the head tilt clearly.

Vision was 20/20 in each eye; the refractive error +1.50D. sph. O. U.

Muscle balance: With the head held upright there was a noncomitant exotropia of 8$^{\Delta}$ with a somewhat variable right hypotropia of 8$^{\Delta}$ at 20 ft., and exotropia of 15$^{\Delta}$ and right hypotropia of 8$^{\Delta}$ at 13 in. Fusion could be attained by tilting the head down and to the left. Diplopia plotting, with the head held straight and the left eye fixating, showed the right image to be higher in the primary gaze, with the vertical separation of the images increasing when the eyes looked down and to the left. There was questionable fusion in other directions of gaze (fig. 3A).

Diagnosis: Exotropia with overaction of the right superior oblique, producing right hypotropia (head upright) and extorsion of the right image.

The operation herein described was performed. Two months postoperatively the patient reported complete comfort. In direct gaze the head was held upright; no diplopia was present. Esophoria of 1$^{\Delta}$ at 20 ft., and exophoria of 4$^{\Delta}$ at 13 in. were noted. The diplopia field showed a slight elevation of the right image with the eyes down and to the left (fig. 3B).

Case 2. Miss E. S., aged 26 years, had a strabismus of long standing, despite a previous operation; she could not read for any length of time or do work of any type requiring prolonged use of the eyes, and noticed diplopia when tired. With the head straight there was a vertical separation of images, the right image being higher. This could be temporarily corrected by tilting the head to the left and down.

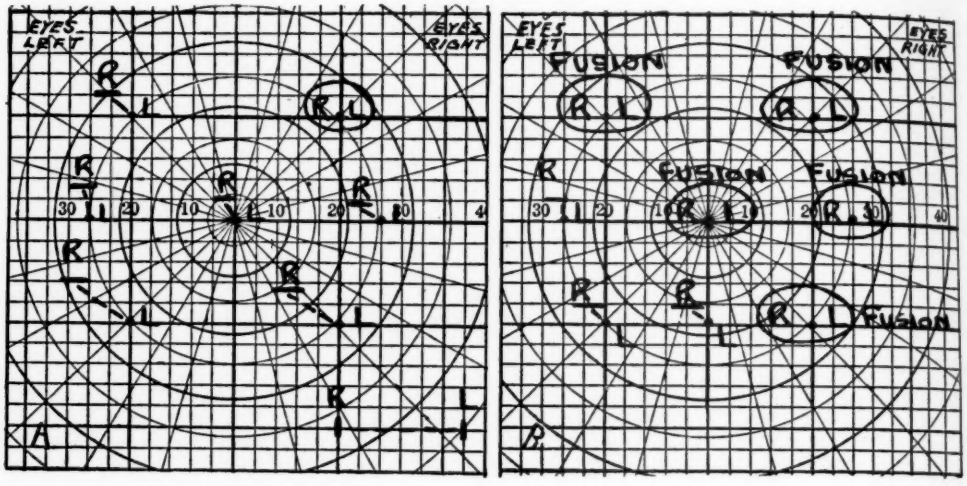

Fig. 3 (Hughes and Bogart). Muscle plotting, case 1: A, preoperative, showing the increasing vertical separation in the lower nasal field or field of primary action of the right eye (left side of the plotting is the patient's left field), and the increasing horizontal separation (secondary action of right superior oblique) in the right field. O.S. fixation. Used at 30 in. B, postoperative, showing fusion over most of the field of motility with a slight residual diplopia in the lower left field. O.S. fixation. Used at 30 in.

Vision was 20/20 in each eye. The refractive error was: O.D., +0.75D. sph. $\bigcirc$ −0.87D. cyl. ax. 12°; O.S., +1.50D. sph. $\bigcirc$ −0.75D. cyl. ax. 175°.

Muscle balance: With the head held upright, a noncomitant exotropia of 6$^{\Delta}$ and a variable right hypotropia of 4$^{\Delta}$ to 6$^{\Delta}$ were noted at 20 ft., and 2$^{\Delta}$ of esotropia and 4$^{\Delta}$ to 6$^{\Delta}$ of right hypotropia at 13 in. An extorsion of the right image of 12$^{\Delta}$ was found on the synoptophore. With the head tilted left and down, and with effort third-degree fusion was temporarily possible for short periods. Examination for aniseikonia in this manner was made and no size difference of the retinal images was found. Diplopia plotting with the head held upright was done, showing diplopia in all positions of gaze, with the right image higher than the left. The

vertical separation of the images was greater with the eyes looking down and left (the left eye was fixating).

Diagnosis: Postoperative exotropia (convergence excess?) and overaction of the right superior oblique.

The right trochlea was recessed in the manner described. The preoperative diplopia fields are shown in figure 4A, as taken during this examination. Postoperatively the patient was followed for 10 months, and reported complete comfort for the first time in years, being able to carry out normal secretarial work. In direct gaze, with the head held upright, there was an exophoria of 10$^{\Delta}$ with 3$^{\Delta}$ of left hyperphoria (with the Maddox rod over the right eye) present at 20 ft., and an exophoria of 5$^{\Delta}$ with 3$^{\Delta}$ of left hyperphoria at 13 in. Residual extorsion of the right image of 3 to 4 degrees was found, when using the synoptophore. The plotting of the postoperative diplopia field is shown in figure 4B. Third-degree fusion could be maintained easily with the head held normally, and near work could be done easily for long periods.

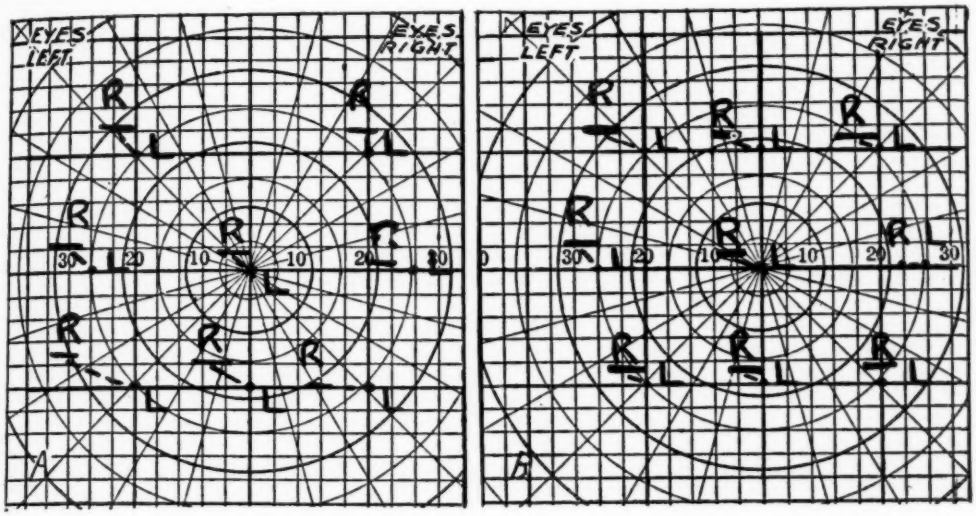

Fig. 4 (Hughes and Bogart). Muscle plotting, case 2: A, preoperative, showing noncomitant variable diplopia. O.S. fixation. Used at 30 in. (Left side of the plotting is patient's left field.) B, postoperative, showing the marked reduction in the vertical separation and noncomitancy with quite constant findings. A small vertical prism was worn to correct the remaining nearly comitant hyperphoria. O.S. fixation. Used at 30 in.

Exact amounts of retroplacement cannot be given at this time due to the fact that sufficient cases have not been recorded.

The advantage of attacking the overactive superior oblique muscle itself is obvious. A study of the plottings will reveal that comitancy of binocular motility was much better following this procedure than could be expected from any of the other procedures recommended for this condition.

Summary

A simple effective means of reducing the effect of an overactive superior oblique muscle is reported. No previous mention of such a procedure has been found in the literature.

LYMPHOGRANULOMA-VENEREUM LESIONS OF THE EYES*

CRISTOBAL ESPÍLDORA, M.D., AND WALDEMAR E. COUTTS, M.D.
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Lymphogranuloma venereum is a general disease. Ravaut, in 1922,[1] was the first to suspect the fact and establish it on the basis of clinical evidence and data that were known and available at the time. In 1933[2] we proved this fact and in 1936[3] demonstrated that this disease was probably homologous to syphilis, for we were able to establish three distinct periods in its development; namely, primary, secondary, and tertiary. The eye and its adnexa may be the site of lymphogranulomatous lesions during any of the aforementioned periods, as we shall endeavor to demonstrate.

EXPERIMENTAL EYE LESIONS IN ANIMALS

Hansmann,[4] in 1924, inoculated a rabbit's eye with pus from an inguinal bubo; the inoculation was followed by panophthalmitis and destruction of the eye. In the same year Gamna[5] inoculated the anterior chamber of rabbits' eyes with lymphogranulomatous material from an enlarged lymph node, but failed to induce lesions. Levaditi and his co-workers[6] infected monkeys by inoculating the cornea and the eyeball with the virus. The corneal inoculation did not result in the appearance of keratitis, but inoculation into the anterior chamber was followed by partial opacity of the cornea, signs of iridocyclitis, and latterly atrophy of the globus ocularis. Phylactos,[7] after experimenting in the same field, concluded that it was rarely possible to inoculate the virus of lymphogranuloma venereum into the cornea of the rabbit; when successful it produces a diffuse parenchymatous keratitis. Satani[8] transplanted into the cornea and anterior chamber of rabbits' and guinea pigs' eyes fragments of testicle from experimentally infected rabbits. Opacity of the media followed, and inclusion bodies, granulo-corpuscles of the Miyagawa type, could be observed in the endothelial cells of Descemet's membrane. In 1936, Levaditi, Schoen, and Reinié[9] inoculated lymphogranulomatous material subconjunctivally into a chimpanzee's eye and noted a marked reaction similar to Parinaud's oculoglandular syndrome.

McNie[10] produced keratitis and uveitis in the eyes of 13 (68 percent) out of 19 guinea pigs by injecting infected mouse brain emulsion into the anterior chamber. Smears from the iris and posterior surface of the cornea of infected guinea-pig eyes, when stained with the Giemsa method, showed structures of varying size resembling the elementary and initial bodies of trachoma.

Findlay,[11] in 1933, noted that after intracerebral inoculation of monkeys with lymphogranulomatous material the pupils were dilated, anisocoria was sometimes present, and there was a loss of coördination of the extraocular muscles of the eye. D'Aunoy, von Haam, and Lichtenstein[12] mentioned briefly the appearance of a mono- or bilateral hemorrhagic conjunctivitis in intracerebrally infected mice and consider this as a prognostically unfavorable sign. Von Haam and Hartwell[13] observed acute hemorrhagic or purulent conjunctivitis in mice, monkeys, ferrets, cats, sheep, and calves after intracerebral inoculation of the animals with the virus of lymphogranuloma venereum, and considered the appearance of these lesions as a positive sign of infection. It

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* In collaboration with the Eye Clinic of Prof. C. Espíldora and the Department of Social Hygiene, Chile Public Health Service, Prof. W. E. Coutts, Chief.

is usually followed by fatal meningoencephalitis.

Cottini[14] infected white mice intracerebrally with lymphogranuloma venereum and, four to five days after inoculation, observed the appearance of a conjunctivitis, catarrhal at first, later purulent. In smears of the discharge he found granulo-corpuscles.

Eye lesions in human beings

Since the identification of lymphogranuloma venereum as a nosologic entity by Durand, Nicolas, and Favre, in 1913, clinicians from different parts of the world have occasionally pointed out the coexistence of eye lesions with certain syndromes of this disease. As knowledge regarding this new infection became widespread and more definite, the aforementioned facts were awarded their true significance and importance, and to such an extent that certain manifestations that appear in the eye are, at present, considered as pathognomonic of the disease.

Eyelids. Our present knowledge as regards lymphogranulomatous lesions of these structures is very meager. In 1936, Weidman and Hunter[15] reported the case of a 35-year-old Negro with lymphogranuloma venereum, who had a marked puffiness of the eyelids for at least five years. This cutaneous change, according to these authors, was associated with the patient's aforementioned disease.

Recently Santos, of Espildora's Eye Clinic, submitted for investigation of inclusion bodies a fragment of a surgically removed chalazion. In sections stained with Mann's classic method we found large numbers of extracellular microcorpuscles very similar in appearance and distribution to the "elementar-Körperchen," described by various investigators (Melczer, Coutts and Martini and others) in lymphogranulomatous tissues.

Conjunctiva. In 1927, Köppel,[16] and in 1929, Hellerstroem,[17] each reported a case of phlyctenular conjunctivitis in man; the lymphogranuloma venereum in Köppel's case was accompanied by severe arthritis.

In 1932 Morax, Nida, and Marteret[18] and Morax and Bollack[19] considered the possibly lymphogranulomatous nature of certain conjunctival granulomas, but abandoned the idea; the Frei test in the patients they studied was negative.

In May, 1936, Hashimoto[20] declared his belief that Parinaud's disease is an infection of the eye with lymphogranuloma inguinale. He had recently seen two cases in which surgeons incised inguinal buboes and soon afterwards there developed a granulating, intensely hyperemic swelling of the conjunctiva. There was a slight swelling of the regional lymph glands. An antigen was prepared from pus from the eye and tested on 10 lymphogranuloma venereum patients, with positive results in all cases. Inoculation of mice with pus from the eye also proved positive.

In the same year Levaditi and his collaborators presented before the June meeting of the Societé Française de Dermatologie et de Syphiligraphie[21] a patient with lymphogranuloma venereum whose primary complaint was an infection of the eye. The patient had a follicular conjunctivitis of the left eye; both lids were edematous and thickened. The Frei test was intensely positive. A petrous regional swelling of lymph nodes was present.

In 1937 Ichijo[22] reported two cases of Parinaud's oculoglandular syndrome in patients with a positive Frei test. In 1938, Hashimoto and Tamata[23] described three cases of Parinaud's disease studied by them up to that date; the lymphogranulomatous nature of the affection was confirmed by animal inoculation of pus from the human eye. In the same year Appelmans[24] described a most conclusive case

in a physician who accidentally infected himself with the virus of lymphogranuloma venereum and developed a conjunctivitis, followed by swelling of the preauricular, submaxillary, parotid, sternocleidomastoid, and subclavicular lymph nodes. Repeated Frei tests gave positive results. In 1939 Ouwejan[25] reported three cases of oculoglandular syndrome with positive Frei tests.

Sonck,[26] in analyzing the occurrence of solar dermatitis in lymphogranuloma venereum pointed out the coexistence of this phenomenon with a conjunctivitis of short duration. Hopkins[27] presented to the Section on Dermatology of the New York Academy of Medicine a case of chronic conjunctivitis which was probably caused by the virus of lymphogranuloma venereum. The first outbreak occurred in 1932, in the right eye with edema of the eyelids, inflamed conjunctiva, swollen and tender ipsilateral auricular and submaxillary glands. The complaint at the time of presentation was localized in the same eye, and the changes were similar to those noted during the first attack with the addition of a purulent discharge. The Frei test was strongly positive.

Curth and his co-workers[28] reported a case of chronic conjunctivitis of seven years' duration and demonstrated the existence of the virus by tissue culture.

In 1930[29] we demonstrated the presence of intracytoplasmic granulo-corpuscles in some surgically removed pterygia.

When pterygium is analyzed from the epidemiologic and clinical points of view, it will be observed that this is a lesion of adult life that occurs more commonly in certain countries and in these in certain regions. It usually appears among the lower and poorer classes of society as is the case with esthiomene, penoscrotal elephantiasis, and other lymphogranulomatous syndromes. Castané Découd[30] mentioned the opinion of Poncet, who considered pterygium as an infectious disease produced by a virus. All accepted this theory.

In our country it is very common among inhabitants of our northern provinces, lying between parallels 18° and 26°, where lymphogranuloma venereum is very frequently observed, especially in and around the ports of Iquique, Tocopilla, and Antofagasta.

Vasquez Barrière[31] of Montevideo, where lymphogranuloma venereum is not uncommon, over a period of 11 years (1930-40) recorded, among 48,960 patients examined at his clinic in the Medical Faculty, 436 cases of pterygium (0.89 per cent) : 184 in women and 252 in men. He pointed out that among 350 patients suffering from lymphogranuloma venereum whom he examined with May he had never observed a pyterygium.

In seven (78 percent) of nine cases of pterygia, surgically removed, we observed inclusion bodies, such as have been found by several investigators (Melczer, Coutts and Martini, and others), in other lymphogranulomatous lesions, when sections were stained by Mann's classic method.[32] One of the patients in this series visited the Clinic five months after the operation with a return of the pterygium; he was operated on again, and inclusion bodies were again found in the stained sections.

Lymphogranulomatous lesions of the conjunctiva may represent early or late manifestations of the disease.

Direct inoculation through the conjunctiva can be compared in more than one sense to primary conjunctival infection by syphilis, Chagas's disease, or American leishmaniasis and tularemia. The inoculation is of the nonvenereal type and the most common means of infection would be the fingers.

The first symptom may be the chancre, which in the special case of lymphogranuloma venereum is usually very small (mi-

cro-chancre) and fugacious. In other cases there may be a simple inflammation of the conjunctiva: a simple conjunctivitis similar in character and nature to other primary manifestations of the disease occurring at the more usual sites of inoculation (urethritis, vulvitis, vaginitis, rectitis). Invasion of the regional lymph node is a common concomitant, rendering it of a hard, petrous consistency, seldom suppurative.

When all the aforementioned symptoms are found to coexist, the clinical picture is that of Parinaud's oculoglandular syndrome. There are, however, forms in which the different symptoms may be very mild, some even absent, as has been observed in abortive forms of the disease (chancre without adenitis, adenitis without urethritis or chancre).

Primary symptoms usually disappear in a short time, but in certain cases, as also when the site is the vagina (chronic ulcer), rectum (chronic rectitis) and elsewhere, the conjunctivitis may follow a chronic course and various lesions may appear in succession.

Chronic conjunctivitis may be stubborn, proliferations may appear, thickening or pterygia may develop, or there may be an involvement of the orifice of the ductus lacrimalis which results in a narrowing or stricture of its initial portion. Such a stenosis is only a repetition of the phenomenon observed in the urethra, rectum, colon, and ileon, when attacked by the virus of lymphogranuloma venereum.

Miculicz's dacryoadenopathy may be of a lymphogranulomatous nature; some authors may previously have thought of this possibility but we have not found any references in the Cumulative Index consulted up to the year 1940. Recently we have studied two lacrimal sacs that were surgically excised and in one of them found inclusion bodies of the microgranular type. Brieva, when completing the histologic study of this sac, found: "Considerable hyperplasia of the epithelium of the sac, which in parts is vacuolized. Intense inflammation, in parts purulent with telangectasis of the subepithelial connective tissue that surrounds the lacrimal epithelium. This inflammation becomes less intense in the zones farther removed from the epithelium and there is an iscrease of collagenous fibrosis."

In regard to the lymphogranulomatous lesions of the conjunctiva, it is of interest to remark that Broadhurst and her collaborators, whose investigations had been limited, on the whole, to the search for inclusion bodies in the epithelial tissues of the upper respiratory tract, also observed their existence in the eye and the genital area.[33] Two subjects who were throat carriers of these bodies showed the same organisms in smears taken from the lining of the lower eyelid; one of these patients was being treated for a chronic low-grade infection of the eyes.

The morphologic appearance of these inclusion bodies is analogous to that found in lymphogranulomatous tissues and smears from vagina and rectum of patients infected with lymphogranuloma venereum. We[34] searched for these inclusion bodies in 30 normal adults and found them in five. The Frei test was positive in three of this latter group of patients, only one of whom had previously suffered from bilateral inguinal buboes, diagnosed at the time as Nicolas Favre's disease. Their presence in apparently normal persons may be associated with the possible existence of extragenital virus carriers.[35]

Cornea. McNie[10] described a mixed form of keratoconjunctivitis in a patient with serologic syphilis and anal fistula with positive Frei test. The cornea showed numerous small areas which stained with fluorescein. Iris and pupillary reactions were normal. Preauricular lymph nodes

were also normal. Vasquez Barrière and May[36] recently described a new syndrome which affects the cornea and may or may not be accompanied by changes in the eyegrounds. According to these writers, it consists in an abnormal visibility and tortuosity of the nerve filets in the central portion of the cornea. Such changes were investigated in 120 patients suffering from acute or chronic forms of lymphogranuloma venereum and were found in a considerable proportion. These writers do not offer an explanation for this phenomenon but limit themselves to pointing out the possible relationship between these findings and those of Ionesco-Mihaesti, such as peri- and endoneuritis with loss of myelin of peripheral nerve filets and other nerve changes, registered in experimental cases of lymphogranuloma venereum.

Malbran and Lima[37] could not confirm the existence of the corneal nerve changes described by the Uruguayan authors. In three patients with lymphogranuloma venereum they found that the cornea had lost its normal transparency and that the stroma, as seen under the optic section, appeared slightly lactescent.

We searched for the corneal-nerve changes described by Vasquez Barrière and May[36] in 20 patients who have recently come under our observation. Our findings are tabulated in table 1.

Among the 20 patients studied in the chart, 12 presented one or another lymphogranulomatous syndrome; the remaining 8 suffered from genito-urinary diseases of diverse etiology. All these cases were studied under the Zapski-Zeiss corneal microscope and the ocular tension was measured with the Schiötz tonometer.

In five cases of lymphogranuloma venereum in two of which granulo-corpuscles had been observed in the lesions, we found an abnormal visibility of the corneal nerves. This symptom was also positive in two cases of nonlymphogranulomatous patients.

Corneal nerves were also studied in patients from neurologic and medical clinics who were sent for eye control or study, and we were able to establish that in a large proportion of these, without clinical or biologic antecedents, the corneal syndrome of Vasquez Barrière was positive.

Based on the aforementioned facts we feel, for the time being, inclined to award this symptom a lesser diagnostic value than that attributed by the Uruguayan investigators.

Vasquez-Barrière and May[36] insist on the diagnostic importance in lymphogranuloma venereum of ocular hypotension, which is commonly (87.5 percent) recorded as under 15 mm. Hg. We have found hypotension in 50 percent of our lymphogranuloma venereum patients, but this phenomenon has also been noted by us in about the same proportion in patients with other diseases and no clinical or other evidence of lymphogranuloma venereum.

Malbran and Lima[37] have also been unable to establish the importance of hypotension as a diagnostic criterion; as regards the abnormal visibility of corneal nerves they have not seen any such phenomenon in lymphogranuloma venereum patients.

Eyegrounds. Funakawa,[38] in 1934, was the first to point out the existence of eyeground changes in relation to lymphogranuloma venereum. In 22 persons who were considered to be normal he found a thickening and tortuosity of the blood vessels near the papilla, the existence of small comma-shaped vessels penetrating the retina behind the central blood vessels, and a radiating striation visible in the nerve fibers around the papilla also attracted his attention. In 10 (45.4 percent)

of these patients he found a positive Frei test, a fact which, in the absence of other positive information, made him attribute these manifestations to lymphogranuloma venereum.

In the same year confirmatory papers on eyeground changes in lymphogranuloma venereum were published by Kitagawa[39] and Espildora and Coutts.[40] Kitagawa reported 37 cases of inguinal lymphogranuloma. The eyegrounds were examined in 30, and peripapillary edema and tortuosity of the blood vessels were noted in almost every case. The cerebrospinal fluid was examined in all these cases: in almost all of them the pressure was abnormally high. The Wassermann reaction was positive in the fluid in three cases and the Meinicke reaction in six. The goldsol reaction was positive in almost every case.

Our first series, studied in 1934,[40] comprised 12 cases: 10 lymph-node syndromes, 1 ano-rectal syndrome, and 1 esthiomene; we reported changes similar to those observed by the Japanese authors.

In a series of 100 cases studied and controlled by us for Chaigneau,[41] we found the following changes: In 92 cases of inguinal lymphogranuloma venereum, there were lesions, in our opinion[3] corresponding to the secondary period of the disease; in 44 (47.8 percent) positive eyegrounds; in 17 (18.4 percent) the changes were doubtful; and in 31 (33.6 percent) the fundi were negative. In eight cases of esthiomene (tertiary-period lesions) five (62.5 percent) were positive, two doubtful, and one negative (specially in normal persons and others suffering from various diseases).

TABLE 1

OCULAR FINDINGS IN 20 CASES OF LYMPHOGRANULOMA VENEREUM

Patient	Diagnosis	L.V. Inclusion Bodies	Cornea		Eyeground		Ocular Tension	
			R.E.	L.E.	R.E.	L.E.	R.E.	L.E.
							mm. Hg	mm. Hg
M.R.	Adenitis L.V.		+	+			14	14
L.M.	Adenitis L.V.	+	+	+	I	I	12	12
O.D.	Adenitis L.V.		−	+	I	II	12	10
L.A.G.	Epididym. L.V.	+	−	−	−	−	21	mm.
P.P.	Epididym. L.V.	+	+	+	−	−	16	14
P.R.	Epididym. chronic	−	−	−	−	−	16	16
E.R.	Epididym. L.V.	+	−	−	I	I	16	16
J.L.P.	Epididym. tbc.		+	+	I	I	10	10
E.A.	Epididym. tbc.	−	−	−	I	I	25	25
S.L.	Phymosis		−	−	II	II	14	14
J.P.	Varicocele		+	+	−	−	12	12
J.S.	Genital tbc.		−	−	I	−T	14	14
A.C.	Eleph. p. et Scr.	+	−	−	II	II	9	10
A.M.	Urethral fistul.	+	−	−	I	I	24	24
O.A.	Rectitis L.V.	−	−	−	I	I	17	18
M.P.	Rectitis L.V.	−	+	+	−	−	12	12
E.U.	Buco-genital L.V.		−	−	III	II		
E.D.	Epididym. L.V.	+	−	−	I			
A.B.	Adenitis L.V. acute		−	−	II	II	10	12
M.G.	Adenitis L.V. acute		−	−	I	I	18	18

These and other findings, specially with regard to the various degrees of involvement exhibited by the syndrome, that were observed in some cases in one or the other eye, the time of appearance, disappearance, and reappearance during the course of the disease, and the absence of subjective symptoms in these patients were also noted in the aforementioned paper. Nakano and Nakasawa[42] confirmed these findings.

In 1937, Vergara,[43] in eight patients

with ano-rectal syndromes and positive Frei tests, found changes in the papilla and in the blood vessels. In five, these symptoms were present in both eyes.

Vasquez Barrière and May,[36] and Malbran and Lima[37] have lately published very interesting findings, most of them confirming those of earlier writers.

Our review of cases, only of the tertiary-period patients (with positive Frei tests) which were observed between 1937-1941 (first six months) may be consulted in the following table:

Grade II. The same aspect of veins and papilla; peripapillary edema.

Grade III. As in grade II, with a pronounced papillitis or papillary edema and changes in blood-vessel crossings.

The symptoms ascribed to our grade I may be liable to erroneous interpretation, and personally we prefer in some cases to report negative or doubtful eyegrounds and repeat the examination twice or oftener at varying intervals. But grades II and III are so evident that no doubt is possible; on several occasions we have

TABLE 2

EYEGROUND FINDINGS IN 140 CASES OF LYMPHOGRANULOMA VENEREUM

Diagnosis	No. Cases	Eyegrounds		
		+	±	−
Rectal syndr. (men)	25	22	1	2
Rectal syndr. (women)	14	8	1	5
Esthiomene	42	38	1	2
Elephs. penis et scroti	10	9	—	1
Elephs. vulvae	6	4	—	2
Urethral stricture	16	11	3	2
Sclero-cystic oöphor.	8	4	—	4
Epididymitis	16	10	2	4
Parametritis	3	2	—	1
	140	108	8	24

Total (71 percent) and partial positive percentages in this series correspond to our former findings and those noted by other investigators.

Ophthalmoscopic changes reported as observed in the eyegrounds were not always present with equal intensity. They may be listed as follows: (a) dilatation, tortuosity, and blackish color of the papillary veins; (b) papillary hyperemia and retinal edema around the optic disc; (c) intense edema or papillitis; (d) changes in the arteriovenous blood-vessel crossings.

We have classified the aforementioned changes according to three grades.

Grade I. Dilatation, tortuosity, and intensely dark color of the papillary veins. Papillary hyperemia.

met with such eyegrounds in patients sent for examination for different causes or who have even consulted us for glasses, and invariably when examined or questioned as regards lymphogranulomatous lesions we have been able to establish a relationship between cause and effect.

The question now arises: "What explanation can be offered for these changes?" Two hypotheses may be formulated and have been considered of importance by various authors.

The first attempts to explain the changes as due to intracranial hypertension, which would find a clinical basis in the fact that headache is a constant symptom in the initial stages of the disease, according to Coutts,[42] in 37.5 percent of the cases. Increase in the spinal-fluid ten-

sion was established in 30 (81 percent) out of 37 cases by Kitagawa;[39] May[36] found 100 percent hypertension with suboccipital puncture. Chaigneau,[43] studying the pressure in 9 cases of our series of 100,[41] found either normal or low values, but in no case hypertension.

The other hypothesis has been proposed by Vasquez Barrière and May, and is cited by Malbran and Lima.[37] They believe that ocular hypotonous is responsible for the retinal-papillary changes. Our personal experience tends partially to confirm their opinion, for we have observed cases in which there coexisted hypotonus with grades I, II, or III of positive eyegrounds. But we must remark that it is not uncommon to find positive eyegrounds with low tension, and low tension with negative eyegrounds.

Sclera. In 1932, Koppel, Liebreich, and Gottlieb[44] reported cases of episcleritis in patients showing severe generalized infection with the virus of lymphogranuloma venereum.

Iris. In 1937, Běhcet[45] described a new syndrome in adults whose three principal symptoms were: aphthoid ulcerations of the mouth and genitalia, accompanied or unaccompanied by iritis. In 1939, in the description of a case of this syndrome, he[46] found in the right eyeground the following changes: the edges of the papilla were blurred and there was intense congestion. The left eye presented congestive neuritis, the blood vessels of the retina were slightly engorged, the vision was normal. This syndrome was attributed by the author to the action of a virus.

Benedek and Olkon[47] recently reported a case of lymphogranuloma venereum in which simultaneously with the appearance of ulcerative skin lesions the patient developed an iritis that was attributed to the virus of lymphogranuloma venereum. This iritis reappeared three times in connection with the recurrence of the ulcerative skin lesions.

In 1934[48] we drew attention to the coexistence of lichenoid lesions of the tongue and genitalia due to the virus of lymphogranuloma venereum. In 1940[49] we summarized two cases seen by us in 1939, presenting iritis and aphthoid lesions of the tongue and penis. Both had intensely positive Frei tests but negative eyegrounds.

This year (1941) we saw two more cases, one with the symptom triad already described, and positive Frei test and eyegrounds. The last case that we have studied was brought to us by Dr. Jimenez; it was characterized by lichenoid lesions of the tongue and penis, a positive Frei test, a grade-III eyeground in the right eye, and a grade-II in the left. Nerve filets were not visible.

In some cases of vitamin-B–complex deficiency (ariboflavinosis), lesions of the eye (keratitis), of the lips (keilitis), tongue (glositis), and of the skin (localized erythemas) may be present, but the Frei test and the eyegrounds are negative.

The relation between this syndrome complex and lymphogranuloma venereum is being studied by us, for many of its manifestations have much in common with this disease, as has been noted by us in a number of papers.

Conclusions

(1) The eye may be the site of lymphogranuloma-venereum lesions during any one of the periods of this disease.

(2) Any structure of the eye may be involved by the process.

(3) All structures in direct contact with the exterior may serve as the site of inoculation for the virus of lymphogranuloma venereum.

Santa Lucia 232.

Vicuña Mackenna 35A.

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EPITHELIAL TUMORS OF THE LIMBUS*

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Consistent with the experience at other points in the body where there is sharp differentiation of epithelium, tumors are found to be relatively common at the limbus. As a reflection of the embryology of the conjunctiva and cornea, epithelial tumors of the limbus are predominantly of epidermal type, and inclusions of epidermal appendages are relatively common, occasionally including elements from the mesoderm and recognizable as mixed tumors or teratomas. Among the pathologic specimens at the Museum there are two such tumors, one involving the cornea. There are 10 dermoids and 5 epithelial cysts. So far as our follow-up records reveal there have been no recurrences after the tumors were excised. Inasmuch as dermoids of the limbus are usually extrophias and not cysts, it is surprising, when one considers how irritating the hairs in such tumors must be, that in several instances treatment has not been sought until the patient has reached the second or third decade of life. The simple epithelial cysts, which naturally are less irritating, were all removed from adult patients. There are in the Registry isolated instances of lymphangioma, xerophthalmia, tyloma, keloid, xeroderma

TABLE 1

DISTRIBUTION BY SEX AND RACE

Diagnosis	Sex		Race		Total
	Male	Female	White	Colored	
Epidermalization and dyskeratosis	13	4	16	1	17
Papilloma	23	5	24	4	28
Carcinoma	42	6	43	5	48
Total	78	15	83	10	93

TABLE 2

DISTRIBUTION BY AGE

Age Group	Epidermalization and Dyskeratosis	Papilloma	Carcinoma	Total
Age 2 years		1		1
20–29	1	1	3	5
30–39	1	3	1	5
40–49	3		5	8
50–59	5	6	11	22
60–69	2	10	13	25
70–79	2	5	10	17
80–89		2	2	4
90–97			2	2
Unknown	3		1	4
Total	17	28	48	93

TABLE 3

INVOLVEMENT OF THE CORNEAL EPITHELIUM

Epidermalization and Dyskeratosis	Papilloma	Carcinoma
2	Histologic 10	19
3	Clinical 4	11
Total 5	14	30

* From the Registry of Ophthalmic Pathology at the Army Medical Museum. Read at the meeting of the American Academy of Ophthalmology and Otolaryngology, at Chicago, October, 1941.

pigmentosa, and lymphosarcoma. These and melanoma of the conjunctiva will not be discussed.

This study is based on 93 pure epithelial tumors arising at or near the limbus and usually coinciding with the palpebral fissure. Eighty-three of the patients are white persons and only 10 are Negroes (table 1). There is also a marked preponderance of males over females; 78 males and 15 females (table 1). This may be an indication of the etiologic importance of chronic irritation. The highest incidence occurs in patients between the ages of 50 and 80 years (table 2), and in only one case did the tumor occur in a patient under the age of 20 years: this was in a child of two. The oldest patient was 97 years of age. The recent work of Phillips[1] in which he reports a great increase in the incidence of skin cancer in the southwestern part of the United States, is of considerable interest. On the surface, our collection would appear to support his findings, as 26 of the 93 cases included in our series are from Texas. The remainder of the patients, including 15 from Tennessee, which rates second to Texas, are distributed among 25 other states, the Panama Canal Zone, and Hawaii. It is felt, however, that so far as our material is concerned, conclusions on the basis of geographic distribution are not justified because our largest contributions of ocular-adnexa biopsy specimens are from Texas and Tennessee. In eight instances, four papillomas and four carcinomas, the tumors

Fig. 1 (Ash and Wilder). Epidermalization and dyskeratosis, corneal epithelium. ×7. Negative No. 72104.*

Fig. 2 (Ash and Wilder). Papillary squamous-cell carcinoma, grade I, with epidermalization and corneal involvement. ×7. Negative No. 72106.

* All illustrations are from Army Medical Museum files.

Fig. 3 (Ash and Wilder). Squamous-cell carcinoma, grade III, with epidermalization and corneal involvement. ×2½. Negative No. 72112.

appeared at the site of and immediately following injury. The series includes a number of cases in which the tumor is of large size, involving not only the limbus but much of the cornea (table 3). There is a great tendency for changes in the limbic epithelium to extend into that of the cornea (figs. 1, 2, 3, 4). Metaplasia of the corneal epithelium is demonstrated histologically in 31 of the 93 cases, and in 18 additional instances there was clinical history of corneal involvement. Rarely is there extension through the sclera into the ciliary body and iris (fig. 5).

Among these epithelial tumors are 17 cases of epidermalization of the limbus epithelium in which there has been dyskeratosis and in which the changes were of sufficient degree to lead to the suspicion of tumor and to excision. In 13 cases the picture is essentially that of leukoplakia (fig. 7) as seen in other mucosas, acanthosis, hyperkeratosis, dyskeratosis of the epidermis, and, in varying degrees, chronic inflammatory reaction in the subepithelial tissue. In four cases the picture approximates that of Bowen's disease of the skin in which there is less acanthosis and more diffuse dyskeratosis. In a number of instances the dyskeratosis in both types is sufficiently bizarre to justify the dermatologists' expression "carcinoma in situ" (figs. 6 and 9). This condition has been seen predominantly in the older-age groups. Only one patient was under 40 years of age. So far as our follow-up

Fig. 4 (Ash and Wilder). Squamous-cell carcinoma, grade I, epidermoid. Epidermalization of corneal epithelium. ×5. Negative No. 72108.

records go there has been no instance of recurrence after simple excision (table 4).

While in the aforementioned group actual neoplasm was not present, epidermalization of adjacent epithelium with or without dyskeratosis was seen in 12 of the 28 papillomas and 35 of the 48 frank carcinomas (figs. 9 and 10). We must conclude that the epidermalization is an important precursor to the development of tumor, be it benign papilloma or carcinoma. There is some basic factor—for example, vitamin-A deficiency, drying, or chronic irritation—that is responsible for this metaplasia, and malignancy is secondary. When we speak of "involvement of the cornea" in one third of the cases it means advancing metaplasia of corneal epithelium and not extension of the actual limbus tumor.

Of the 28 papillomas, 25 consisted of epidermal type of epithelium, and in 3 instances conjunctival type of epithelium was retained. Eighteen were treated by excision alone, in which group there was one recurrence (table 4), eight by primary enucleation usually necessitated by extensive involvement of the cornea. In none of the 28 cases diagnosed "papilloma" microscopically has there been clinical evidence of malignancy.

Of the 48 frank carcinomas 39 were of the squamous-cell type, predominantly epidermoid, 8 were papillary squamous-cell types and there was only 1 basal-cell

Fig. 5 (Ash and Wilder). Squamous-cell carcinoma of limbus extending through sclera into ciliary body and iris. No metastasis 5½ years after enucleation. ×7. Negative No. 66879.

TABLE 4

RECURRENCE FOLLOWING EXCISION AND ENUCLEATION

	Excision		Enucleation		Excision Followed by Recurrence and Enucleation	Total
	Recurrence		Recurrence			
	+	−	+	−		
Epidermalization and dyskeratosis	1	14		2		17
Papilloma	1	17		8	2	28
Carcinoma	2	23		19	4	48
Total	4*	54		29†	6*	93

* Ultimate cures.
† One died of possible metastasis, 1½ years following enucleation.

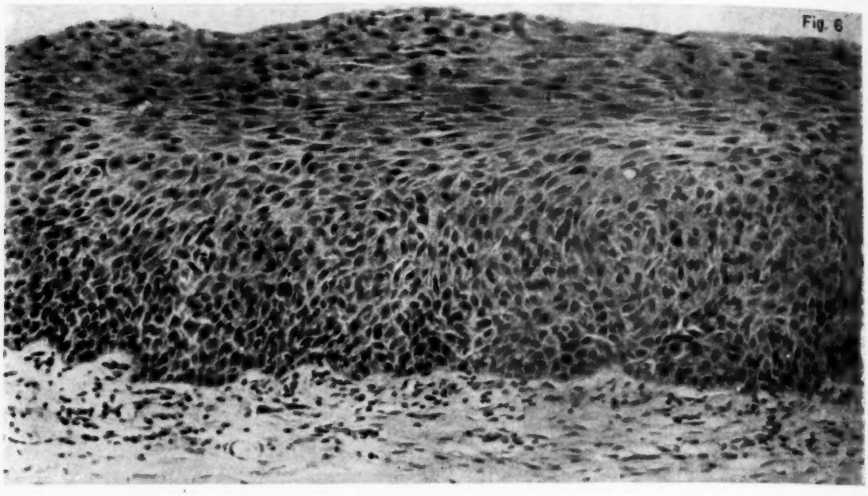

Fig. 6 (Ash and Wilder). Epidermalization and diffuse dyskeratosis, Bowen type. "Carcinoma in situ." ×110. Negative No. 72109.

type. Simple excision completely eradicated the tumor in 23 instances, in 2 instances additional excision was necessary, and in 23 cases enucleation was performed. In only 1 patient of the 48 was death attributed to the tumor and that only presumptively. It would seem that primary conservative measures, therefore, followed by enucleation if the recurrence is persistent, are justified, and that there appears to be no necessity for pre- or postoperative irradiation.

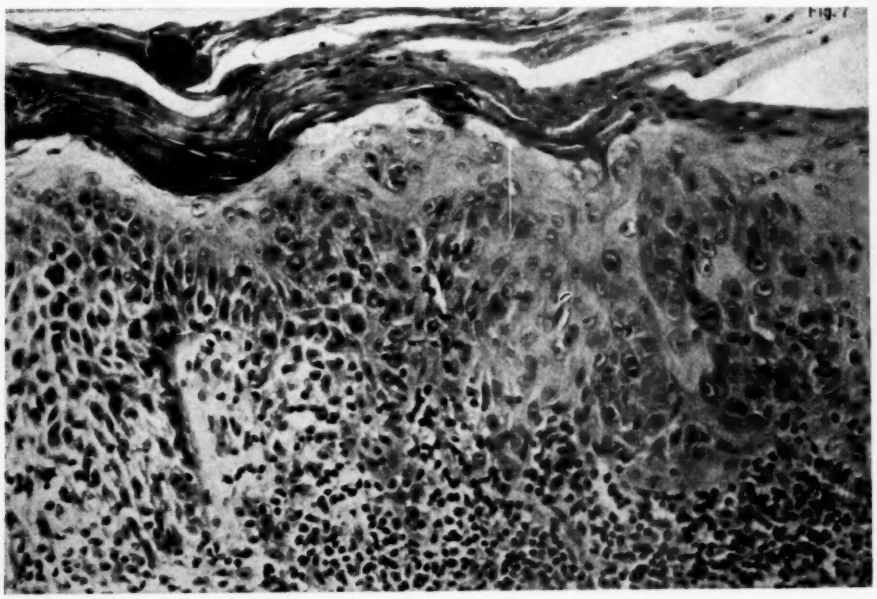

Fig. 7 (Ash and Wilder). Epidermalization, hyperkeratosis, dyskeratosis, leukoplakic type, and chronic inflammatory reaction. ×175. Negative No. 72086.

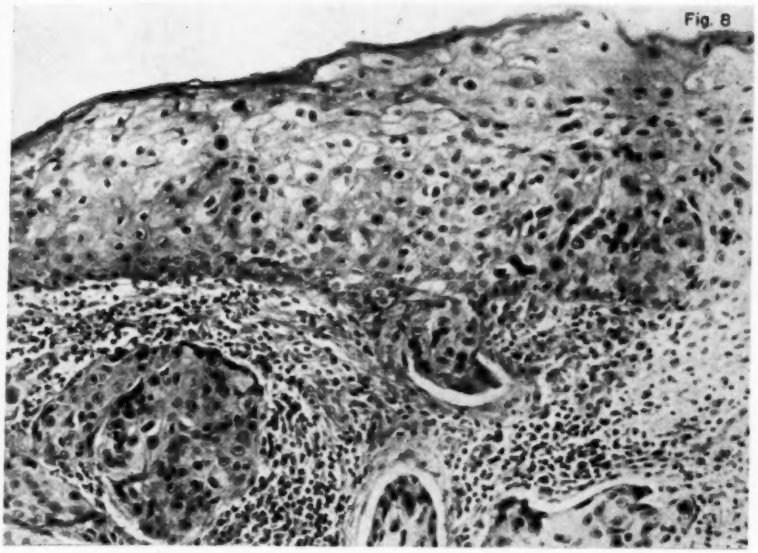

Fig. 8 (Ash and Wilder). Epidermalization and dyskeratosis, similar to that in figure 6, but with malignant invasion of subepithelial stroma. ×110. Negative No. 72111.

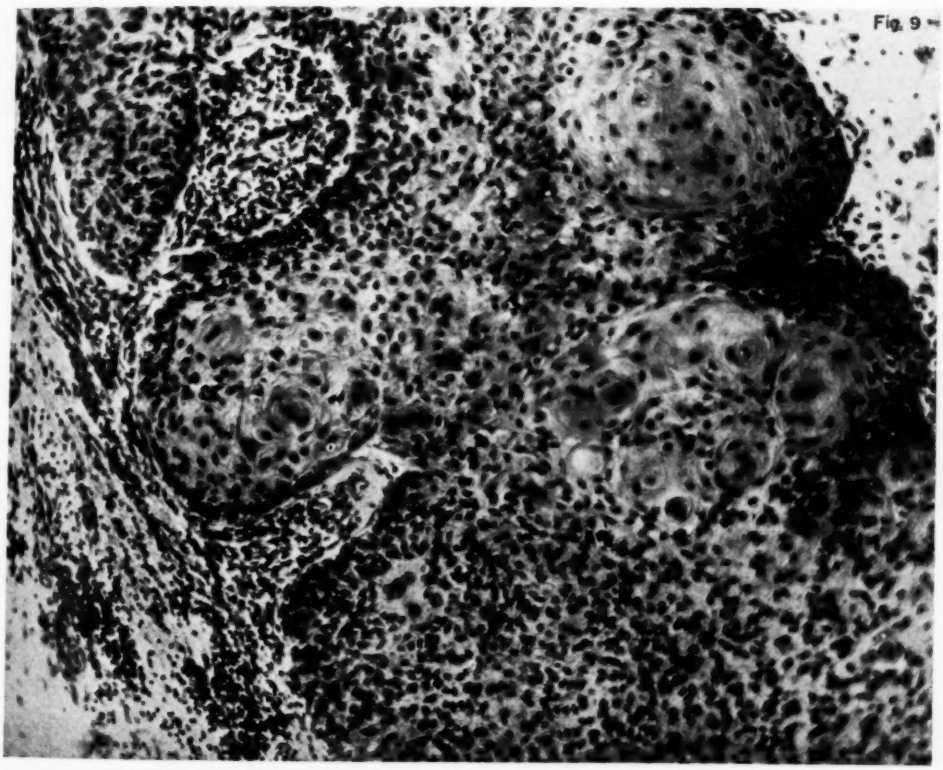

Fig. 9 (Ash and Wilder). Squamous-cell papilloma with malignant dyskeratosis. Another example of "carcinoma in situ." ×110. Negative No. 63167.

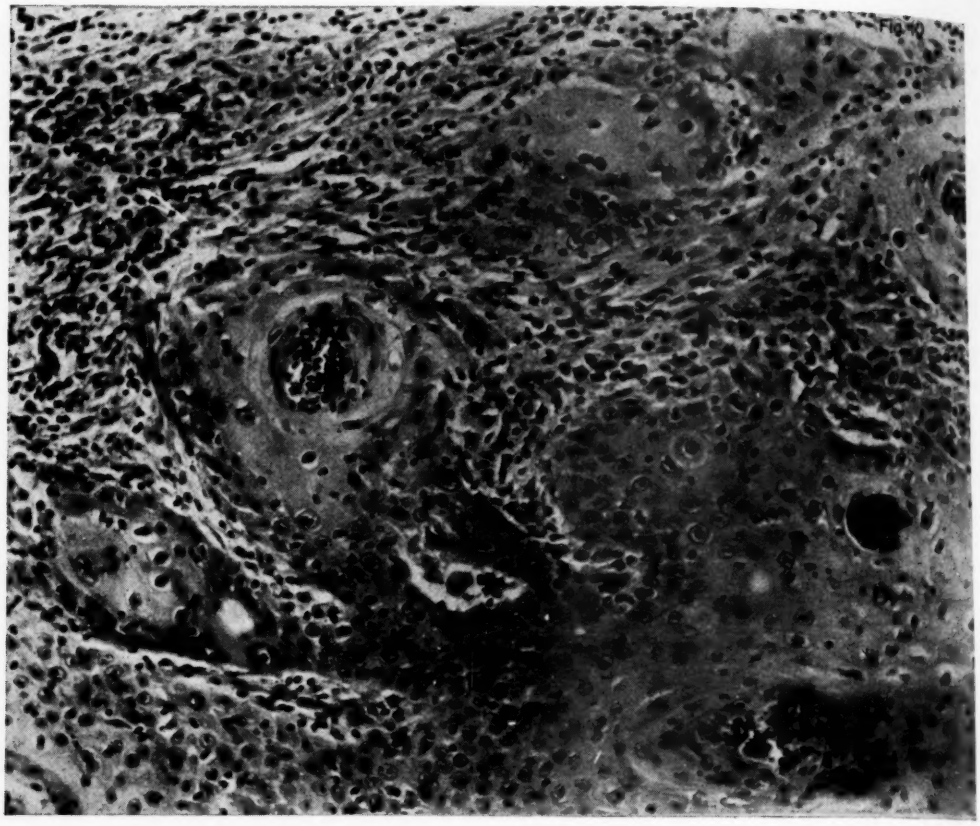

Fig. 10 (Ash and Wilder). Frank epidermoid carcinoma occurring in a papilloma. ×150. Negative No. 72094.

Conclusions

1. Epithelial "tumors" of the limbus in adults are frequently simply leukoplakic metaplasia of conjunctival epithelium.

2. Papilloma or squamous-cell carcinoma may develop secondarily in this altered epithelium.

3. Basal-cell carcinoma is rare at the limbus.

4. The tendency is to involvement of the corneal epithelium rather than to extension peripherally.

5. Penetration of the sclera and metastasis are rare even in large carcinomas.

6. The pathologic diagnosis of "carcinoma" is not necessarily an indication for irradiation in connection with radical surgery. Enucleation of the eye appears to be justified only where there is already appreciable involvement of the cornea, or after local excision has failed.

If one must have a cancer, the limbus would seem to be as safe a place as any to have it.

Reference

[1] Phillips, C. Skin cancer in southwest United States. Study No. 1. A statistical survey of 1,482 cancers in Texas. Third Intern. Congr. on Cancer, Atlantic City, N.J., 1939, Sept., p. 32.

TETANUS FOLLOWING EYE INJURY

Report of case: Review of literature

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My attention was drawn to the possibility of tetanus following injury to the eye by the occurrence of a case in my own practice. Never before having witnessed such a phenomenon, I undertook some research and found that tetanus as a complication of eye injuries is of very rare occurrence, no more than 30 cases, apparently, having been reported up to the present time (1941).

As the last publication concerning tetanus as a complication of eye injury appeared more than two years ago (see footnote, p. 944), it has seemed worth while again to review the literature, in connection with the publication of my case, bringing the total number now reported to 31.

Case report

A farmer, aged 39 years, came under my care on May 15, 1939. Late in the afternoon of that day, while making repairs to his barn, a nail he was driving ricocheted, striking him in the left eye. Admitted to the hosptial that same evening, preliminary examination showed vision in the right eye to be 6/7.5; left eye 0. There was a perforating wound of the globe, approximately 1 cm. long, situated at the limbus, opposite the 8-o'clock position. The laceration extended a distance of 5 mm. over the surface of the cornea, the iris prolapsing into the wound. The anterior chamber was filled with blood, there was vitreous discharge at the site of the wound in the sclera, and no tactile sensation of tension.

A prophylactic injection of 1,500 units of antitoxin was administered shortly after admission.

Treatment and Clinical course. Under local anesthesia, the prolapsed iris was excised, the wound repaired, and a pressure bandage applied. The next day there was marked local reaction, with considerable edema of the conjunctiva. Foreign protein, in the form of typhoid vaccine, was given intravenously, producing a satisfactory response. Convalescence thereafter was uneventful, the patient being discharged from the hospital on May 23d.

Subsequent history. Four days later, on May 27th, the patient returned saying, "Yesterday I felt fine until late afternoon when my lips began to get thick and felt funny, my hands and feet began to swell, and an eruption appeared on my body. When I got up this morning I could not open my mouth very wide, and it feels now as though it were clamping down."

Upon readmittance and examination, unmistakable evidence of tetanus was found. After a subcutaneous desensitization, intensive tetanus antitoxin therapy was instituted, by muscular, spinal, and intravenous routes, using 375,000 units in all. The outstanding manifestations in order of appearance were: Urticaria, generalized stiffening of all joints, rigidity of the neck, paralysis of the third and seventh nerves, impaired hearing, and generalized adenopathy. On the seventh day of the patient's second hospitalization a stormy convalescence began, and not until June 10th, 26 days after the original injury, was it deemed wise to attempt to remove the left eye. Convalescence from this operation was uneventful, and he was finally discharged from the hospital on June 21, 1939.

Literature

The literature regarding tetanus due to the eye injuries is almost wholly in the French language. Those Americans who fought in Northern France during the first World War, the medical officers in particular, well recall the fearful prevalence of *Bacillus tetani* in a soil where for uncounted centuries the patient Norman horses have contributed to its enrichment. Nearly every case reported provides data relating the eye injury to some direct contact with horses, or with ground presumably contaminated by their excrement. In France, perhaps more than any other country, are circumstances so frequently favorable for the infection of an injured eye with *Bacillus tetani.*

Yet, oddly enough, the first case of which we have record occurred in Ireland! The account, published in the Dublin Medical Press, was cited in a German treatise on eye injuries, and this German article was later abstracted in the French Annales d'Oculistique. As the English text is not available in this country, we are compelled to accept the account as it has filtered down to us through three languages.

This report was made in 1847. The next instance appears to have been in 1854) the third in 1870, and the fourth in 1882. The years 1886, 1892, and 1895 saw each one case reported, while in 1894 there were two. Thus up to the beginning of the twentieth century but nine instances of tetanus following injury to the eye had been cited in medical literature. I wish to emphasize this particularly because the recent infrequency of the occurrence of this complication to eye injury might readily be attributed to the introduction of better prophylaxis, tetanus antitoxin most of all. But comparison of the periods before and after the introduction of antitoxin shows that the condition has always been rare.

In the twentieth century, however, the volume of literature concerning tetanus following eye injuries noticeably increased. Though the actual incidence was no greater, the interest in the subject intensified, especially with the coming of the first World War. From the beginning of the century to 1914, the first year of the war, 10 cases are recorded, and by 1920, when all known cases were tabulated by Castelain and Lafargue,[2] and Maurice Collin[5] gave résumés in his Paris thesis, the total number of recorded instances had risen to 23. Quentin,[11] writing in 1937, had found seven additional cases, one of which had originally been seen as far back as 1915. The most recent writer on the subject, Cogan of Boston,[4] did not add any cases, but rejected one previously included in the list as "probable but unproved." My case would, therefore, be the thirty-first to be placed on record. Brief protocols of these cases follow, most of them being translated from Collin's thesis, as but few of the originals in the earlier literature are available.

Case reports

Case 1 (Pollock, Dublin Medical Press, No. 441, 1847). A man, aged 33 years, was struck in the left eye with a whiplash. The cornea was completely divided, but there was no hernia of the iris. On the sixth day there was purulent secretion in the eye, in the evening trismus; eighth day abundant secretion, paralysis of the facial nerve; ninth day, generalized tetanus and death.

Case 2 (case of tetanus reported from Saint George's Hospital, London. Med. Times & Gaz., 1854, No. 202, p. 488). Man, aged 33 years; wound of the cornea; etiology not given. Panophthalmia; paralysis of the left facial nerve; on sixth day generalized tetanus; death on the tenth day.

Case 3 (Kirchner, Aerztl. Bericht ü.d. könig. preuss. Lazareth in Versailles, 1870. Quoted by Fromaget). Man, aged 33 years; gunshot wound of the right eye. Pronounced exophthalmos due to hemorrhage into the orbit; vision abolished. One the seventh day, paralysis of the right facial nerve; on the eighth day, embarrassment in swallowing; hypopyon; trismus on ninth day, and on the tenth, pronounced tetanus and death.

Case 4 (Becker, Archiv f. Psychiatrie, 1882, v. 12, p. 250). Patient was hit in the eye (right) by a whiplash; panophthalmia on the third day, trismus on the eighth day, the mouth being forced open only with difficulty, and the jaw deviated toward the affected side. Difficulty in swallowing on the ninth day, with complete paralysis of the left common ocular motor nerve (with the exception of the fibers motivating the sphincter of the iris); tenth day complete immobilization of the eye, with edema of the papilla; practically impossible to separate the teeth. On the eleventh day opisthotonos, accompanied by tonic and clonic spasms of the upper extremities, with death on the fourteenth day.*

Case 5 (Ramiro-Guedes, Archiv. Opht. de Lisbon, 1886, No. 4 [26]. Quoted by Aubaret). Injury of left eye from whiplash, making a vertical wound in the cornea. On the second day, exophthalmos and hypopyon; on third day, trismus and opisthotonos. Recovery after one month, but with eventual phthisis bulbi.†

Case 6 (E. G. Rust,[12] Jour. Ophth., Otol. & Laryngol., 1892, v. 4, p. 357). A woman, aged 45 years, was struck in the eye by the metal arc of a toy bow. As neither the iris nor the crystalline lens was injured the wound was not regarded as serious, but an iritis developed, with manifestations of tetanus on the eighth day, and death on the tenth day. The author remarks that he had never before heard of tetanus following an eye injury.

Case 7 (Chevalier, Thèse de Bordeaux, 1894). This is merely a citation of a case seen by Lannelongue, where the elder Denucé had performed an operation on the eye, which was followed by tetanus and the death of the patient.

Case 8 (Fromaget, Arch. d'Opht., 1894, v. 14, p. 657). A boy, aged 19 years, was struck in the eye by a metal spindle, rupturing the cornea and the sclerotic, evacuating nearly the entire contents of the globe. The eye was washed with water from a nearby well. On the fourth day he experienced difficulty in opening his mouth, but he was not admitted to Professor Badel's service until the tenth day when a pronounced trismus was in evidence. The injured eye was removed, but on the twelfth day opisthotonos developed, with tetanic convulsions on the fourteenth day and death on the nineteenth day.

Case 9 (Schnitzler, Wiener klin. Rundschau, September 9, 1895). A man, aged 48 years, was struck by buckshot in the right eye, producing panophthalmia on the third day. Not until the twelfth day, however, did he complain of acute pain on mastication. On the fourteenth day there were trismus and paralysis of the right facial nerve, but no rigidity of the neck muscles. Hospitalized, the lower half of the injured cornea was found to be necrotic; on incision pus exuded. On the uninjured side, the left upper lid could be lifted only with difficulty, the pupil was contracted and did not react to light, while the eye movements were much limited especially above (paralysis of the third and sixth pairs of nerves). The mouth could not be opened, and contraction of the masseters and muscles of the pharynx was in evidence. The orbit was exenterated, and the wound cauterized. In the globe after removal was found the shot which had caused the injury and when this was bacteriologically examined it was found to harbor tetanus bacilli. That evening the neck became rigid, and on the night following the sixteenth day there were many convulsions; ptosis of the remaining eye increased, and death took place on the seventeenth day.

Case 10 (Goering, Inaug. Diss. Halle a. S., September, 1903; also cited by Schmidt-Rimpler). A boy, aged 12 years, was struck in the left eye by a fragment of porcelain soiled with earth, causing a perforating wound of the cornea, through which the iris prolapsed. The iris was excised, followed by panophthalmia, necessitating exenteration. On the sixth day trismus, with complete paralysis of the left side of the face. The right eyelid could only be lifted with difficulty. On the evening of the ninth day opisthotonos with tetanic convulsions. On the tenth day the uninjured eye could not be closed and paralysis of the right external rectus was in evidence. No tetanus bacilli could be found in the secretion of the wound. Antitoxin was administered on the eleventh day, and, after a slight relapse, with one or two mild tetanic manifestations, he was discharged as cured.

Case 11 (Mauch; quoted by Groenow, section on Tetanus, Graefe-Saemisch Handbuch, v. 2, section 22). A clot of earth caused lesions to the cornea, followed by tetanus and death on the tenth day.

Case 12 (Lépine; quoted by Worms, Thèse de Lyon, 1905). A man, aged 49 years, underwent enucleation of his right eye because of panophthalmia caused by the blow of a whiplash. On the sixth day it was difficult to open the mouth and movements of the remaining eye were difficult. On the eighth day the masseter muscles remained somewhat contracted but mastication and deglutition were not inter-

* Collin believes this to be the same case reported by Schultze in Neurologische Centralblatt, 1882, No. 6.

† This is the first recovery noted, but, without access to the original account, I have been unable to verify it.

fered with. Hospitalized on the ninth day, the right facial nerve was found paralyzed, with horizontal wrinkles in the forehead which the patient could not control, but without vertical wrinkles. On the left, movements of the eye were limited to abduction, and there was almost complete ptosis. The pupil did not react either to light or in accommodation. All the facial lines on the left side were exaggerated, the labial commissure was raised and widened, the nasolabial fold accentuated, but it was difficult to demonstrate actual contractures, or to decide if the facial aspect were due to a right-sided paralysis. The mouth could be freely opened, but when an attempt was made to explore the pharynx, contractions were excited. The tongue was movable, though deviated to the right. On the tenth day the patient complained he could not open his jaws, while facial paralysis and ptosis were more pronounced. On the eleventh day the patient was unable to swallow, every attempt at deglutition provoking pharyngeal spasms. Trismus was in evidence. Antitoxin was administered and chloral hydrate given. The injection appeared to provoke generalized convulsions, which increased on the thirteenth day, when the neck became rigid. On the fourteenth day ophthalmoplegia and complete ptosis of the left side of the face; death on the fifteenth day.

Case 13 (Oller, Arch. f. Augenh., 1905, v. 51, p. 121). Patient's right eye injured by penetration of a stable fork. On the third day the eyelids were much swollen and chemosis pronounced. The cornea had a vertical tear, the edges of the wound being infected, and the entire cornea was of a grayish-yellow color. The débris of the iris had prolapsed into the wound. The globe protruded and was almost immovable. The left eye was intact. Surgical intervention was refused at this time. On the fourth day the swelling of the eyelids had extended over the entire right side of the face, and on the sixth day the tip of the nose and the corner of the mouth were drawn toward the left side, probably because of the extreme engorgement of the right side. The following day the asymmetry was so pronounced that facial paralysis was diagnosed. The patient ground his teeth but was still able to open his mouth, but by the eighth day there was marked contraction of the masseters, but no rigidity of the neck, nor opisthotonos. The same afternoon, tetanus being recognized, the right eye was enucleated, but death occurred the following day. At autopsy it was found that the sclerotic had been perforated as well as the cornea, and a blade of grass was found in the globe and another under the eyelid.

Case 14 (Gerock; cited in Klin. Monatsbl. f. Augenh., 1906, v. 45, p. 114). A man, aged 46 years, was struck in the eye by a whip, making a penetrating wound of the sclerotic, and causing panophthalmia. The initial symptoms of cephalic tetanus appeared on the seventh day, whereupon the eye was enucleated. In it was found a shred of the whiplash, about seven centimeters long. Despite antitoxin injections the man expired on the tenth day.

Case 15 (Körnigshoffer; cited by Klin. Monatsbl. f. Augenh., 1906, v. 45, p. 114). A boy developed panophthalmia after being shot in the eye by an arrow. The eye had to be enucleated and on the sixth day there were manifestations of tetanus. Despite the use of antitoxin the boy died nine days after the accident.

Case 16 (Pincus,[10] Centralbl. f. prakt. Augenh., 1911, v. 35, p. 353). On December 14, 1909, a workman was struck in the left eye by a whiplash, with instantaneous loss of vision. Entering the clinic on the second day, chemosis was marked and exophthalmos beginning. In the greatly injected eye the iris could be seen herniated through the wound and covered with pus, the hernia being between the limbus and the ciliary body. The hernia was excised and the wound in the sclera sutured. On the third day the swelling of the eyelids, the chemosis, and exophthalmos had increased, and the following day, believing the exophthalmos to be due to a hematoma of the orbit, the eye was removed. On the ninth day swallowing became difficult and the mouth could hardly be opened. On the tenth day there was trismus, facial paralysis on the affected side, paralysis of the common motor ocular, and ptosis on the uninjured side. Though antitoxin was now administered, the rigidity of the neck increased, accompanied by tetanic convulsions, death following on the twelfth day. Examination of the encleated eyeball disclosed a fragment of the whiplash in the wound.

Case 17 (De Rider and Danis, Société belge d'Opht., December, 1911). A man, aged 29 years, was struck in the left eye by a whiplash, causing a horizontal wound in the cornea and part of the sclerotic. Several days later, because of panophthalmia, the eye was enucleated, and a fragment of the lash was found. On the ninth day there was a slight trismus, which was at first attributed to the use of a gag during anesthesia. Later, the tetanus bacillus was found in the secretion from the eye socket. On the eleventh day trismus was marked and antitoxin was administered. There was no opisthotonos at any time, and though there was some rigidity of the muscles of the extremities, the patient eventually recovered.

Case 18 (Aubaret,[1] Arch. d'Opht., 1913, v. 33, March, p. 148). A little girl of seven

years was struck in the left eye by a fragment of reed-grass, which caused a perforating wound of the upper part of the globe. After a few days it was found necessary to enucleate the eye because of generalized inflammation. The reed fragment was found in the removed globe. The day following the operation the little patient had a suffocating attack, and the mouth could not be opened. The remaining (right) eye deviated toward the right and upward, while one side of the face contracted. Antitoxin treatment was at once instituted, but a little later in the same day trismus was pronounced on both sides, with right-sided facial paralysis. Because of tremendous edema on the left side of the face it was impossible to determine whether or not the facial nerve was paralyzed. The choking attacks became more frequent, with all symptoms of tetanus becoming more manifest. Opisthotonos set in, with death the same evening. Examination of the fragment of reed found in the enucleated eye showed tetanus bacilli.

Case 19 (Wirtz, Klin. Monatsbl. f. Augenh., 1905, v. 46, p. 386). A case of panopthalmia following a blow from a whiplash was seen by this author, who, having determined the tetanus bacillus in the secretion from the eye, enucleated the eye, and administered antitoxin, 48 hours after the initial injury. The classic symptoms of tetanus never developed, and for this reason most authors do not include this case in their collections.

Case 20 (Vinsonneau,[15] Arch. d'Opht., 1913, v. 33, p. 418). A boy, aged 17 years, had slightly injured the conjunctiva of one eye by a flying splinter from a tool which he was using to pry loose a piece of wood. The context does not make clear whether the splinter was of metal from the tool, or from the wood which was being pried loose. Though examined at once by a physician, the wound was not regarded as serious and when panophthalmia developed at the end of the second day, it was impossible to find the portal of entry of the foreign body into the conjuntiva and sclera. There was still further delay because the patient was a minor and an employed workman, so permission for operation had to be obtained before the urgently needed enucleation could take place. The account states that chloroform was used as an anesthetic and that the patient took it badly because he was an alcoholic, which seems odd at age 17. However, it was found necessary to prop his mouth open during the anesthetization, and when he had difficulty in opening his jaws the following day no importance was attached to this manifestation, and the masseter muscles were not palpated, nor did the patient complain of a stiff neck during the succeeding 36 hours. That night, however, a frantic "coup de telephone" from the Franciscan sister who was his nurse called the physician to behold all the classic signs of tetanus. Serum was injected subcutaneously, in conjunction with other routine measures to combat tetanus, but again red tape tied their hands and prevented intracranial injections without "authorization." The patient died the following evening, after almost continuous convulsions; there was no autopsy, nor examination of the secretions from the injured eye, which the author remarks he "very greatly regretted."

Case 21 (Schneider, E. R. [Copenhagen, Denmark], Ann. d'Ocul., 1916, v. 153, p. 395). A coachman, aged 36 years, entered the author's service almost two weeks after being struck in the left eye by a whip. Examination showed that the cornea was ruptured, with hernia of the iris; chemosis was pronounced and the cornea already necrotic; there was slight exophthalmos and the globe was immovable, while the neighboring parts, the cheek especially, were swollen and infiltrated, and drooping of the corner of the mouth indicated paralysis of the facial nerve. The patient could not unclose his jaws, although the neck was not rigid and there had been no convulsions, so the diagnosis of tetanus was not made. Under chloroform, made difficult because this patient also was an alcoholic, the swollen tissues were incised. Because no pus was found it was decided to exenterate the globe. No foreign body was discovered, and the posterior portion of the sclerotic remained intact. A simple dressing was applied, but the next day trismus, facial paralysis, and slight convulsions made plain the true condition. Antitoxin was then vigorously pushed, with other antitetanic measures, but despite a slight improvement the following day, the patient expired, approximately 17 days after the accident. At autopsy no foreign body was found, and there was only slight infiltration of the retrobulbar tissues.

Case 22 (Goetz [patient of Professor Rollet[6]], Thèse de Lyon, 1916). The patient was a veterinary surgeon, aged 30 years. While cutting a piece of iron with a saw, a fragment of the metal lodged in his eye. There was little pain at first, but the next day the pain became so severe he visited Professor Rollet's eye clinic, where the cornea was found to be necrotic with chemosis of conjunctiva and anterior chamber. Application of a magnet caused severe pain. Iridectomy and incision of the globe permitted the foreign body to be drawn out by the magnet. Six days later trismus developed, with rigidity of the neck, slight dysphagia, and left facial paralysis. As soon as the symptoms of tetanus appeared the eye was enucleated and antitoxin administered.

The patient survived for a week, with no opisthotonos at any time, the convulsions being limited to the upper part of the body, the head muscles especially. Death occurred 18 days after the accident.

Case 23 (Castelain and Lafargue, Ann. d'Ocul., 1920, v. 157, January, p. 9). An artilleryman, brought by ambulance, was found, on reaching the base hospital, to have a panophthalmia, due, he explained, to a scratch from a splinter of iron which had entered his eye while he was hammering upon a horseshoe. He was sure the fragment had not been retained in the eye, but radiographic examination showed the fragment lodged in the upper part of the globe. As it was routine for surgeons at the front to give injections of antitoxin to all wounded, the question as to whether or not he had received antitetanic treatment was not asked of the patient. The following day the eye was exenterated, the fragment extracted, and the patient referred to a civilian ophthalmologist, who saw nothing unusual until the tenth day after the accident. On that day the left side of the face showed partial paralysis, while the right side was sharply contracted, the patient breathed with difficulty and could hardly open his mouth. The neck and arm muscles were not contracted. Vigorous antitetanic measures were at once instituted, and despite trismus and difficulty in deglutition, the condition somewhat improved, and by the eighth day all symptoms were much ameliorated. A relapse took place on the thirteenth day, but thereafter the eye symptoms regressed while the facial paralysis remained the same. On the thirtieth day the mouth could be normally opened, and the patient was able to eat and drink without difficulty. By the fortieth day all symptoms had subsided save a slight trace of facial paralysis.

Case 24 (Colin, Maurice,[15] Thèse de Paris, 1921). An artilleryman entered the base hospital with an erosion of the left conjunctiva, caused by one of his companions-in-arms striking him in the face with a broom. The left eye was badly bruised and the conjunctiva on the lower outer section of the globe showed a slight scratch. The following day there was an intense conjunctivitis, with the eye vessels greatly congested, and the condition remained stationary despite all treatment. Four days after the accident trismus set in, the patient complaining he was unable to chew his food, although there was no pain in the jaws. During the following night, the tetanic symptoms were exaggerated, and at the same time painful contractions began, the jaws being firmly locked. A chloral enema could not be introduced because of spasm of the sphincter, but a hypodermic of morphine somewhat relieved the spasms. Antitoxin was administered on the fifth day, instillations being made directly into the eye. The condition however, became steadily more grave, the convulsions were violent and intense, the paroxysms being no longer limited to the face but involving the thorax also. This greatly embarrassed respiration, and eventually caused death by paralysis of the respiratory muscles, the end coming on the third day after the début of the tetanic symptoms.

Case 25 (Sattler, Robert,[13] Arch. of Ophth., 1918, v. 47, p. 64). A farmhand, aged 21 years, while seated in the back of a wagon, had his right eye perforated by an arrow made of dried "horseweed," shot from a toy bow by a boy standing on the roadside about 15 feet away. The patient jerked out the arrow with his own hands (very likely contaminated with the excreta of horses) and at once sought medical aid. Foreign material was removed from the eyeball, but whether of earth or of the dried weed, the patient could not tell. Admitted to Cincinnati General Hospital 13 hours after the injury, the wound was found to be limited to the cornea, the iris prolapsing through it. When it was learned "that the improvised arrow had just been pulled out of the ground by the boy who shaped and shot it," it was deemed wise to enucleate the eye at once. For three days following operation convalescence was normal, but on the third day the patient complained of a stiff neck and inability to swallow, the right side of the jaws being especially affected; examination showed trismus already established. Intensified antitetanic treatment was at once instituted; the orbit was exenterated, and serum applied locally to the wound, as well as being administered by subcutaneous and spinal injections. The patient however, expired on the seventh day after admission.*

Case 26 (Berger, A. H., Inaugural dissertation at Würtzberg, 1927). This case is mentioned by both Quentin and Cogan, but the author has been unable to get access to the original, or to find any abstract in ophthalmologic literature. Cogan mentions that some injuries were due to "firecrackers," and as no mention of this is found in the articles available for abstracting, it seems likely that firecrackers were the etiologic agent in this case. It is also mentioned that Berger recommended high dosage of antitoxin, and the application of antitetanus serum direct to the wound.

Case 27 (Jacqueau and Bujadoux,[8] Lyon méd., 1925, v. 136, p. 442). A man aged 25 years, was wounded by a piece of rusty iron

* This appears to be the second case of tetanus following eye injury to be reported in English.

wire, which perforated his left eyeball. Admitted immediately to the hospital, he was given an intravenous injection of colloidal gold, and 5 c.c. of milk intramuscularly. This treatment was repeated on the two days following, but on the morning of the third day an emergency enucleation of the eyeball was carried out "before the infection had opportunity to extend further." The immediate convalescence was satisfactory, but on the fourth day, when healing had progressed far enough to permit the fitting of an artificial eye, the complete syndrome of cephalic tetanus was presented, trismus on the left side, and facial paralysis, most marked on the right at the level of the orbit; there was no pain at first. Antitetanic serum was at once administered, 40 c.c. daily for five days; then 20 c.c. daily until 400 c.c. had been used. Chloral was given to control the convulsions. Treatment had no apparent effect for four days, but thereafter the patient began to mend, and, being sustained by a liquid diet until the rigidity of the jaws relaxed, eventually made a complete recovery.

Case 28 (Stark, Adolf,[14] Klin. oczna, 1929, v. 7, pp. 82-83 [Polish]; Abst. in Zentrlb. f. d. ges. Ophth., 1929-30, v. 22, p. 854). A man, aged 42 years, was rendered entirely blind in one eye when it was struck by the lash of a horsewhip. After one week the eye began to suppurate and the cornea became necrotic. The initial symptoms of tetanus were spasm of the glottis with inability to swallow, left-sided facial paralysis, and trismus. The eye was immediately enucleated and antitetanus treatment instituted, but the patient died on the eighth day.

Case 29 (Jacqueau,[7] Bull. Soc. d'Opht. de Paris, 1935, p. 452). A boy, aged 14 years, who was hunting out moles with the aid of a "pistolet" especially designed for the purpose, caused an explosion which threw earth into his face. The left eye was filled with foreign material and so injured that vision was abolished. Seen on the third day there was no facial paralysis, but there was much conjunctival edema and a yellowish opacity in the center of the globe, together with all the evidences of a beginning panophthalmia. A radiograph revealed a foreign body in the "yellowish" area. Though an immediate enucleation seemed wise, because the infection appeared still latent, operation was delayed for a few days "to permit the child's mother to accustom herself to the brutal decision," that is, removal of the eye. But on the eighth day after the accident facial paralysis appeared, with difficulty in swallowing and a light trismus. The eye was immediately enucleated and in the globe a fragment of stone was found. High dosage of antitetanic serum was instituted without delay, but for several days thereafter the classic manifestations of tetanus were in no degree ameliorated. Trismus was so marked that for two weeks the child could take nourishment only through a straw passed between the fast-locked teeth. There was great rigidity of the neck muscles and almost complete ptosis of the remaining eye. Pneumonia also increased the complications, reducing the little patient to a skeleton, but after two weeks improvement began, and all manifestations disappeared save a severe tachycardia, the pulse being too rapid to count. This eventually subsided, however, so the cure was complete.*

Case 30 (Quentin, G.,[11] Bull. mem. Soc. Franc. d'Opht., 1937, v. 50, p. 92). A stonemason, aged 55 years, while mixing mortar, felt a foreign body lodge in his right eye. He paid no attention to it at the time, and when questioned later was unable to tell under what circumstances the injury was received, save that it occurred while he was "at work." Seen by the ophthalmologist after two days, the foreign body could be made out almost in the center of the globe, surrounded by a small zone of infiltration, of the whitish-yellow hue. The conjunctiva was hyperemic and there was even a little perikeratitic injection, yet the condition was regarded as trivial because there was no synechia and not the least trace of hypopyon. Vision was reduced to 1/15 and the pain was now so great that sleep was impossible. The foreign body was easily extracted, the eye irrigated by way of the readily permeable lacrimal ducts, and the edges of the ulcer cauterized with galvano-cautery. The wound was dressed for three days, at the end of which period the ulcer had extended considerably and hypopyon appeared. Treatment was continued as before and in three more days hypopyon had diminished, all evidence subsiding by the sixteenth day, when the patient was allowed to go home. He did not report daily as ordered and was not seen again for three days, when his wife assisted him to the clinic, showing marked trismus, quite unable to open his mouth, and complaining of a "sensation of curvature" which caused his body to assume the classic position associated with tetanus. Immediate antitetanic treatment, pushed to the limit of safety, had no effect for several days, the condition growing progressively worse. Not until 23 days after the accident was there any improvement, though the condition of the eye wound remained as upon discharge from the hospital. On the twenty-third day the patient was able to chew and swallow, and within two more days to walk unaided, though his gait

* In discussion of this paper, two further cases were cited by Aurand and Genet, but no details were given, so it is impossible to include them in the tabulation.

was unsteady and jerking. The temperature, which had reached 38.1°C., gradually returned to normal, and on the thirty-second day after he was first seen, the patient was discharged, fully cured.

Etiology

The cause of tetanus following eye injury is, of course, contamination of the wound with the *Bacillus tetani,* known in France as the "Bacillus of Nicolaier." Therefore, the agent by means of which the bacillus entered the eye becomes of chief etiologic importance. In nine cases this agent was a horsewhip, a fragment of the lash being found in the wound when the eye was enucleated in cases 14 and 16. In case 13 the injury was caused by a stable fork, and in case 24 by a broom, presumably one being used about a stable, as the patient was an artilleryman in the days when cannon were still moved largely by equine power. Another artilleryman was hammering a horseshoe from which a splinter flew into his eye. Thus 12 cases were due to agents known to have been in direct contact with horses, while splinters of wood, or metal, a bit of rusty wire, a nail from the barn roof (author's case), a farmhand's, and a veterinary's hands, which are mentioned in 5 more cases, may very well have been contaminated by similar contact. Thus 17 cases, or almost 60 percent, can be traced to a source well known as highly favorable to *Bacillus tetani* infection.

In three instances the cause of the injury was known to have been contaminated with earth, and in several others the likelihood of such soiling was very great. The firecrackers, toy arrows, and pieces of weed and grass must have inevitably come in recent contact with the ground. None of the authors whose cases are listed have stressed the importance of contaminated hands coming in contact with the wound, though in one instance it is mentioned that the eye was washed with water from a nearby well. Only one case followed operation on a previously uninjured eye, and, because we have been unable to read this in the original, it is impossible to state whether or not this operative wound also may have been in some way exposed to the excreta of horses, or contaminated earth.

The actual amount of injury does not seem to have any relation to the liability to tetanus. Some of the wounds were very serious, deep tears in the globe, hernia of the iris, and extensive destruction of the conjunctiva. Others were mere scratches, so trivial that neither patient nor physician considered them important enough to take any precautions against grave consequences. This emphasizes the wisdom of such precautions under any and all circumstances. Most of the patients were adults who suffered injury in the course of their ordinary occupation, and none were "war wounds" in the proper meaning of that term, the one bullet wound having been received under civilian conditions which were not precisely stated.

Pathology

Several authors—notably Castelain and Lafargue, and Aubaret—are of the opinion that unless a panophthalmia takes place, the conditions essential for the propagation of the tetanus bacillus will be lacking. Cogan notes that in 16 cases there is specific mention of panophthalmitis, of hypopyon in three, corneal ulcer in 1, and 1 instance of severe conjunctivitis. In the later cases, including the present author's, routine precautions taken as soon as the patient came under care probably lessened the incidence of such complications. But reports of all early cases mention marked suppuration, which was undoubtedly due to organisms other than *Bacillus tetani* because infections with this bacillus in other parts of the body often do not suppurate at all.

Experiments with rabbits have shown that tetanus more often results from mixed infections than when tetanus bacilli are inoculated in pure culture. In the more recent reports there is usually mention of bacteriologic investigation, resulting very infrequently in recovery of the tetanus organisms, however. In Wirtz's case (no. 19) the bacteria were demonstrated, but clinical tetanus never appeared, antitetanic measures being promptly instituted. For this reason many editors do not include this with the "undoubted" cases.

It is argued by Cogan that it would seem logical to remove the injured eye as soon as any possibility of tetanus is noted, thus lessening the dangers of dissemination. Yet if enucleation is not done *before* panophthalmia develops there would seem to be but little use in operating with this purpose in mind, for the infection will in all probability have already extended beyond the orbital tissues.

However, the records of the seven patients who recovered, including my own, would seem to offer evidence in favor of radical removal. In case 10, panophthalmia followed excision of the prolapsed iris, on account of which the eye was enucleated. This was apparently done immediately, while the symptoms of tetanus were not manifested until the sixth day. In the case of De Rider and Danis (no. 17) the eye was enucleated "several days later" when panophthalmia developed, but tetanic symptoms first appeared on the ninth day. In case 19, the disputed case of Wirtz, the presence of tetanus bacilli was first determined by examination of the secretions, and thereafter, the eye was enucleated and atitoxin administered. Castelain and Lafargue's patient already had developed panophthalmia when they first saw him, so the next day they removed the eye and referred the patient to a civilian ophthalmologist who saw "nothing unusual" until 10 days had elapsed, when facial paralysis developed. In case 27, the eye was enucleated on the third day after the accident to prevent "further spread of the infection." This would indicate a panophthalmia, although it is not specifically mentioned.

The most complete information regarding this point is provided by case 29 where the patient was not seen by Jacqueau, who reported it, until the third day after the accident. At that time there "was evidence of a beginning ophthalmia" but the surgeon did not remove the eye immediately because he wished to accustom the child's mother to the idea of his losing his eye! After waiting eight days facial paralysis appeared, whereupon the eye was removed but without stopping the course of classical tetanic symptoms. The fact that antitoxin in high dosage was employed in all these patients who recovered no doubt influenced the favorable outcome, but as it was used equally in most of the fatal cases, we can draw no very precise conclusions. It is altogether probable that a generalized infective process took place in every instance, even when no specific mention of it was made.

Symptoms

The lapse of time between the initial injury and the appearance of the first symptoms of tetanus varies considerably, but in only a few instances did they supervene rapidly. In case 5, exophthalmos and hypopyon appeared on the third day according to available data. In case 8, where the eye had been washed with water from a nearby well (which was in all probability near a stable, and contaminated with tetanus bacilli), "difficulty in opening the mouth" began on the fourth day though the full syndrome was not witnessed until the twelfth day. Collin reports that trismus began four days after the accident. But in most instances the length of time was greater, the 12 days

in my own case being about the average.

Cephalic tetanus, called by the French *maladie de Rose* or *syndrome de Rose,* is the type of disease regularly seen at the outset in cases where an eye lesion provided a portal of entrance for the organisms. But in the words of Castelain and Lafargue: "In tetanus consecutive to ocular lesions, the disease rarely remains limited to Rose's syndrome; the tetanic infection is not confined to the head in any cases save that of Ridder and our own. Generally three or four days after the appearance of trismus, difficulty of deglutition, and facial paralysis, the tetanus is generalized so as to reach the upper and lower limbs, ending unfortunately in the regular manifestations of tetanic infection. We say *unfortunately,* for a single glance at this clinical picture as set forth in the case reports shows a fatal outcome in most of the cases. The prognosis is grave indeed, and that much the more serious as the onset is precocious."

Trismus, the classic "lockjaw," is the first symptom to call attention to the condition in the majority of cases. Contraction of the jaw muscles causes "gritting" of the teeth, soon followed by stronger contractions which clamp the jaws so firmly together that it is quite impossible to separate them, any attempt to do so causing spasms which only serve to fix them more firmly. Even liquids sometimes fail to pass, and a sudden contraction at times will cause the tongue to be severely bitten when making attempts to introduce nourishment into the mouth. The hard contracted masseter can often be distinctly outlined by palpating the skin above it. When liquids have been passed through the teeth, a spasm of the glottis often prevents their being swallowed. Stiffness of the neck appears to be a less prominent and constant sign in tetanus of ophthalmic origin than in that originally affecting other parts of the body. Facial paralysis regularly supervenes promptly upon trismus, always taking place on the side of the injured eye. In six of the cases collected by Castelain and Lafargue (recorded prior to 1920) there was ocular paralysis also, sometimes in the affected eye, sometimes on the opposite side of the orbit, or again, on both sides at once, or in rapid succession, the common ocular-motor nerve being, in all cases, the one involved. In their own case, as well as that of Schnitzler, not only the common motor ocular, but also the sixth pair, showed paresis.

Of the later cases, that of Quentin had "only a slight paralysis of the right (injured) side of the face," while ocular "palsy" does not appear to have been thought worthy of special record in the others. The other eye symptoms mentioned are all common to tetanus manifestations seen generally, no matter where the lesion of entrance chances to be located. It has already been mentioned that the strictly cephalic symptoms quickly evolve into those associated with the generalized form of tetanus. Besides Castelain and Lafargue's case, only the patient of De Rider and Danis never presented generalized tetanus.

Course and Prognosis

The course which the disease pursued in my case seems to be fairly typical of all the instances of tetanus following eye lesions. My patient was discharged from the hospital on the eighth day after injury with the eye wound in good condition, and no expectation of further trouble. The first symptoms of tetanus appeared on the fourth day thereafter, and the disease ran a characteristically stormy course for six days, after which the severe symptoms abated, until by the fourteenth day, the condition was sufficiently

good to permit removal of the injured eye. It is not possible to compare this to the cases earlier reported where no prophylactic injections of antitetanus serum had been given, as this undoubtedly influenced the course of the disease, and probably the final satisfactory outcome. Cogan, writing in 1939, says, "Prophylactic antitoxin was apparently not used in any of the patients in this series, and there is no report of tetanus having occurred from ocular injuries where it was used." My case, therefore, would seem to be the first report, both of its use and its failure to prevent the occurrence of clinical tetanus.

Cogan goes on to say that the relative infrequency of tetanus from ocular injuries during the first World War can doubtless be accredited to the liberal use of antitoxin, inasmuch as many of those wounds must have been contaminated by ground containing tetanus spores. The nail which strick my patient's eye had not been in direct contact with the ground, but the fingers which had handled it may very well have been contaminated by contact with stable dust. Cogan wrote his paper largely as an argument against the routine use of prophylactic antitoxin in eye injuries, unless the injury has "been caused by objects associated with horses directly, or with ground probably contaminated by manure." He concludes that "it would seem justifiable, therefore, to give antitoxin in those cases where there is a history of such contamination."

Inasmuch as 80 percent of the cases proved fatal, whether or not antitoxin was administered *after* tetanic symptoms appeared, and that my patient, the only one to receive a prophylactic injection, did recover, I feel I am in a position to advocate the routine use of prophylactic antitoxin administration for eye injuries, the same as is now the custom in wounds sustained by other parts of the body. Though ocular tetanus is a very rare occurrence, the extremely high mortality makes any preventive measures justifiable, and, in my opinion, the "dangers" foreseen by Cogan are trifling in view of the value which such measures *may* assume.

In the majority of the recorded cases, death followed very rapidly the first symptoms of clinical tetanus. In 12 of the 17 fatal cases collected by Collin, the end came before the fifth day. He explains the rapid spread of the infection by the fact that the peripheral nerves convey it to the central nervous system, and when the wound of entrance is in the eye, "the extremely rich nerve plexus of the cornea on the one hand, and the proximity of the ramifications of the optic nerve on the other hand, greatly facilitate the attack of the toxin upon the nerve centers."

In the few patients who survived we find that complete restoration to health did not take place for at least a month. My own patient's recovery was quite rapid when compared with that in the earlier cases. Relapses seem to have been fairly frequent, but in the end all the tetanic manifestations disappeared completely without leaving permanent nerve alterations, as might have been expected in view of the severity of the complications.

Summary and Conclusions

A case of tetanus following injury to the eye is repored. Prophylactic antitoxin was administered at the outset, but the classic syndrome of cephalic tetanus supervened notwithstanding. The patient recovered eventually.

Search of the literature revealed that but 30 cases of tetanus following eye injury have ever been reported. In most of the cases the causal factor was an object immediately associated with horses, or with earth which might conceivably have

been contaminated with the excrement of horses.

The mortality is extremely high, only 6 out of 30 patients surviving, making the death rate 80 percent. In most of the cases seen after the beginning of the twentieth century, tetanus antitoxin was administered as soon as clinical signs of tetanus developed. In all recovered cases antitoxin was thus given. Only in my own case was *prophylactic* administration employed.

Inasmuch as recovery has only taken place when antitoxin was used, the advisability of routine injection of such serum after any eye injury would seem to be indicated. Though one should not generalize from a single instance, the case herein reported is offered as supporting evidence for this recommendation.*

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* When the foregoing paper had already been completed and placed in the publisher's hands, there appeared the article by G. G. Jayme of Goyaz, Brazil, entitled "Cephalic tetanus following injury to the eyeball" (Amer. Jour. Ophth., 1941, v. 24, Nov., p. 1281). In this publication the history of the condition is very fully reviewed and many references are cited to which I, unfortunately, did not have access for verification, so could not include them in my bibliography. The Spanish literature, in particular, is meagerly represented in the large medical libraries of the United States. Jayme's paper is of great interest, and should prove a valuable addition to the literature of the subject of tetanus following eye injuries.

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COMPARISON OF CASES OF ATYPICAL AND TYPICAL ACHROMATOPSIA

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INTRODUCTION

Congenital achromatopsia or loss of perception for all chromatic colors occurs much less frequently than does congenital red-green color blindness. The number of reported cases of achromatopsia is, however, sufficient to determine quite definitely the characteristic findings associated with this anomaly. A small percentage of the reported cases are atypical in one or more respects. Because of the rarity of these atypical forms there is considerable disagreement as to their explanation and classification.

The present investigation reports comparative studies of three subjects with achromatopsia. The results in one of these cases correspond in every way with the usual picture, whereas the second and third cases are evidently atypical. The data from all three are presented here with the belief that a comparison of the findings may throw further light on atypical forms of achromatopsia.

HISTORICAL

Typical achromatopsia. An analysis of the cases reported in the literature[1] indicates that the characteristic findings in congenital achromatopsia are as follows:

(1) There is complete inability to perceive chromatic color. The subject is able to match perfectly any region of the spectrum with any other or with achromatic (white) light,† by suitable variation in the quantity of the achromatic or the spectrum light.

(2) Visual acuity is invariably reduced, usually to about 1/10 on the Snellen scale. Other evidences of foveal impairment, found frequently but not invariably, are nystagmus or a history of previous nystagmus, and a central scotoma. Although there are many instances in which a central scotoma cannot be demonstrated, this may well be due to the nystagmus, since the unsteadiness of fixation may be sufficient to prevent detection of even the normal Mariotte blind spot.

(3) There is a shortening of the long-wave end of the spectrum and a shift in the region of maximum brightness toward the short-wave end.

(4) There is marked photophobia with high luminance (photometric brightness).

(5) The inheritance of the defect differs fundamentally from the recessive sex-linked inheritance found in typical red-green color blindness. Achromatopsia tends to occur in a number of individuals in the same generation and occurs with approximately equal frequency in males and females. Consanguinity of the parents of the affected individuals is frequent. The occurrence of achromatopsia in two successive generations of a family is rare. On the basis of these observed facts it is generally believed that achromatopsia is inherited as a simple recessive characteristic.[1-3]

A few studies of the achromatic sensitivity and of the rate of dark adaptation have been made in subjects with typical

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† An effort has been made to employ the terminology which will be recommended in the forthcoming report of the Colorimetry Committee of the Optical Society of America, L. A. Jones, chairman. In some cases an old usage has been placed in parentheses to suggest the meaning of the new term.

achromatopsia, but there is little agreement in the findings. Kondo[4] and Bonnet, Rollet, and Paufique[5] reported that the rate of dark adaptation is the same as that of the normal eye. Rönne[6] and Uhthoff,[7] on the contrary, found the rate of dark adaptation to be more rapid than in the normal eye, while Kravkov[8] found it to be less rapid. May[9] found that when the luminance (photometric brightness) of the adapting field was not so high as to cause blinking or partial closure of the lids, the curve of dark adaptation agreed with that of a normal control subject tested under the same conditions. But if the adapting luminance was higher, the color-blind subject would close his lids partially and so interfere with light adaptation. His rate of dark adaptation under such conditions was naturally more rapid than that of the normal subject.

Hofmann and Nussbaum[10] found that after light adaptation with very high luminance the thresholds of a subject with achromatopsia were higher than normal during the early stages of dark adaptation. Subsequently this result was confirmed by Hecht *et al.*[11] These authors found that the curve of dark adaptation of a totally color-blind subject, determined after previous adaptation to high luminance, was a single continuous curve. Kohlrausch,[12,13] however, was the first to show that under the same conditions the curve of dark adaptation of the normal eye is made up of two independent sections. In the first portion, he believed, the thresholds are determined by cone response because the cone threshold is below that of the rods; in the second portion, the thresholds are those of the rods because the rod threshold is here lower than that of the cones. If cones are absent or functionally defective in achromatopsia, the first section of the adaptation curve should differ from that of the normal eye. The findings of Hofmann and Nussbaum, as well as those of Hecht *et al.*, are in accord with this view. They found that the thresholds in the first part of the adaptation curve were higher for achromats than for normals. Other investigators did not find such an increase in threshold, but in their case the level of previous light adaptation may have been insufficient to ensure dependence of the early portion of the curve on cone response; or, if sufficient, the achromat may have been prevented by blinks, and other interference, from adapting to as high a luminance as did the normal subject.

The first case of achromatopsia in the literature was reported in 1777 by Huddart.[14] In 1926, Julia Bell[1] published an extensive monograph on color blindness that includes a detailed study of the 119 cases of achromatopsia reported in the century and a half following Huddart's paper. The majority of these cases conform closely to the typical picture, the characteristics of which we have outlined above. The majority of the more recent contributions likewise describe cases of typical achromatopsia.[8,15-26]

Atypical achromatopsia. Among the reported cases of congenital achromatopsia there are a small number in which there is either some possibility or definite evidence of deviations from the characteristic picture. Bell pointed out that the subjects in the 119 cases diagnosed as achromatopsia were *assumed* to be able to match all colors to a suitably chosen achromatic stimulus, but that this test was not made in the great majority of the cases. She concluded that it is not possible "to insist upon the totality of the inability to discriminate between different colors in all cases." Furthermore, 4 of the 119 cases furnished definite evidence of achromatopsia associated with normal visual acuity.[14,27-29] In these same cases, neither nystagmus nor photophobia was reported. The luminosity curve of the

spectrum was examined in 34 of the subjects, and there were two who showed no shift in the region of maximum brightness.[28, 30] These two subjects showed achromatopsia in one eye only.

In addition to the small group of atypical cases reviewed by Bell, cases which probably should be classified similarly have been reported by Kondo,[4] Richardson-Robinson,[31] Best,[32] Snyder,[33] Shindo,[34] Heinsius,[35] and Franceschetti.[36]

Snyder reported a subject who confused colors normally perceived as reds, yellows, greens, and grays, but who could discriminate blue. Except for the incomplete loss of chromatic vision, the subject in this case exhibited all of the characteristic findings associated with achromatopsia. Franceschetti reported on four subjects with incomplete loss of chromatic sense who were otherwise typical. Kondo described two siblings who had a weak remnant of color perception, but who were otherwise typical. Richardson-Robinson described a patient with normal foveal acuity in whom the color blindness was complete for blue and yellow and perhaps complete for green; but there was definite evidence of ability to differentiate red.

Shindo made a study of two siblings, one of whom had all the characteristics of typical achromatopsia, while the other had normal foveal acuity and no nystagmus, but otherwise typical achromatopsia. In Best's case there was normal foveal acuity in the left eye. The acuity of the right eye was reduced slightly as a result of pathologic changes in the macular region. The luminosity curve of the spectrum showed no shift toward the short-wave-length end.

Heinsius reported a case of complete achromatopsia in which there was normal foveal acuity and neither nystagmus nor photophobia. Furthermore, the region of maximum brightness in the spectrum agreed with that of the normal eye. The visible spectrum was, however, shortened at both ends as indicated by an observed range of only 460 to 640 mμ.

The question of the inheritance of atypical achromatopsia is unsettled. This seems to be due to the small number of cases available for study and the inadequacy of the information regarding their pedigrees. Podesta[37] has suggested, however, that the atypical forms may follow the laws of sex-linked heredity rather than those of simple recessive heredity. If this is correct, there is support for the view that the atypical cases are more closely related to simple red-green blindness than to achromatopsia.

Anatomical basis for achromatopsia. The typical form of achromatopsia has been variously attributed to complete absence or loss of function of the retinal cones, or to some abnormality in the functioning of the cones. If there were absence or complete loss of function of the cones and they were not replaced by rods in the foveal region, we should expect total blindness at the fovea. Because of the unsteadiness of fixation, however, it seems impossible to determine experimentally whether the fovea is completely blind or only partially so.

The only anatomic investigation of the retina of a subject with congenital achromatopsia was that made by Larsen[38] in 1921. He found the number and distribution of rods and cones to be entirely normal. In the fovea, however, where the cones are normally long and slender, they were found to be short and plump with very short outer limbs. Surrounding the fovea there was a region containing short thick cones interspersed with shorter rods. At some distance from the fovea, the retinal structure appeared to be entirely normal. The clinical findings in this case of Larsen's have been described by Holm and Lodberg.[25] The subject presented the

classical symptoms of reduced central acuity, nystagmus, photophobia, shortening of the red end of the visible spectrum, and shift in the region of maximum brightness. Hers was one of the 23 cases described by Holm and Lodberg; all of these subjects lived in a small community where the isolation and intermarriage would favor frequent manifestation of a recessive disease. There seems to be no doubt, therefore, that Larsen's case was one of typical congenital achromatopsia. The findings indicate that the anomaly may have been due to a defect in the functioning of the cones rather than to their absence.

Defect in the functioning of the retinal cones would provide a better explanation for the classical form of achromatopsia than for the atypical forms. Ladd-Franklin[39] and Heinsius[35] have suggested that those cases of achromatopsia in which there is a normal luminosity curve, normal foveal acuity, no nystagmus, and no photophobia are perhaps attributable to some defect in the conducting pathways or in the higher centers rather than to a defect in the retina itself.

Present investigation

This paper reports detailed studies of two females: A. B., 15 years of age, and E. W., 29 years of age. J. W., a man, aged 23 years, was also examined, but he was not available for a period of time sufficient to complete the series of tests. The findings from the two female subjects will be considered first.

The following groups of tests were given to the subjects A. B. and E. W.: (1) Tests to determine the nature and degree of the defect in chromatic vision. (2) Tests of foveal function. (3) Tests of achromatic sensitivity. (4) Ophthalmoscopic examination of the eye. (5) Study of the pedigree, if known, to determine the type of heredity of the anomaly.

1. Chromatic vision

With the Nagel anomaloscope subjects A. B. and E. W. could match both red and green of spectral purity to yellow, by suitably adjusting the luminance of the yellow. Tests with the Ishihara pseudo-isochromatic charts gave the following results:

Subject E. W.: 12, —, —, 5, 71, —, —, 3, —, 5, —, —, —.
Subject A. B.: 12, 8, —, —, —, —, —, —, —, —, —, —, —.
Normal response: 12, 8, 6, 5, 74, 2, 6, 5, 7, —, —, 26, 42.
Typical red-green color blind: 12, 3, 5, 2, 21, —, —, —, —, 5, 2, 2 or 6, 4 or 2.

The results of the Ishihara test indicate a greater deviation from normal for both subjects than is usually shown in simple red-green color blindness.

A further series of color matches was made on the photometer bench. One of the photometric fields was illuminated by a 100-watt Mazda lamp directly and the other by a 250-watt Mazda lamp transmitting through a Wratten filter. The filters used were numbers: 71A (red), 73 (yellow-green), 74 (green), and 76 (blue). The two subjects differed in their responses to this test also. Subject A. B. was able, by adjusting the relative luminances, to obtain a perfect match between the standard and comparison fields with each of the four filters. Subject E. W. reported that the green and yellow-green appeared slightly yellowish in comparison with the standard. When the blue filter was used, she stated that the perceived blue faded with continuous observation. Similarly, the red comparison field was perceived as a red that soon faded. The fact that E. W. consistently named the red correctly in this test, but had matched a spectrum red to a yellow in the Nagel anomaloscope, can be explained perhaps by the greater field-size in the photometer.

In perimetric studies made with test

objects of red and blue Heidelberg paper, E. W. was able to recognize a 3-degree red stimulus in a small central field extending about 5 degrees or 6 degrees from the point of fixation. A red test object subtending a 1-degree angle was seen as red in a very small field extending not more than 2 degrees from the fixation point. A 0.5-degree red test object was not recognized as red. A 1-degree blue test object was called blue in a field of about 20 degrees extent. The visual fields for both eyes are shown in figure 6.

Although too much reliance must not be placed on the color names given by subjects with proved abnormalities in chromatic vision, the consistency and reproducibility of the findings in this case lead to the conclusion that the loss of chromatic vision for subject E. W. was not complete. A. B., on the contrary, showed in all the tests a complete absence of chromatic vision.

2. FOVEAL FUNCTION

Subject E. W. exhibited no nystagmus; perimetric examination showed no evidence of a central scotoma; and the visual acuity was normal. With moderate luminance, subject A. B. had a visual acuity of 10/70 in each eye that was not improved by correction of the refractive error. There was definite nystagmus also; but the perimetric examination revealed no central scotoma, either relative or absolute. The nystagmus and reduced acuity indicated that the normal superiority of the fovea in moderate luminance was absent. At very low luminances the visual acuity did not differ significantly from that of the normal eye. Acuity measurements were made at five different luminances both for A. B. and for a normal subject under the same conditions. The results are given in table 1 and figure 1. The acuity of A. B. is seen to increase but slightly with increase in luminance.

TABLE 1

ACUITY MEASUREMENTS OF A. B. AND A NORMAL SUBJECT AT EACH OF FIVE LUMINANCES

Luminance of Background (ml.)	Log lum. of Background (micro ml.)	Binocular Acuity	
		A. B.	Normal
17.2	4.236	0.175	1.33
0.416	2.619	0.15	1.0
0.0406	1.608	0.10	0.6
0.0134	1.127	0.088	0.4
0.00390	0.591	0.063	0.2

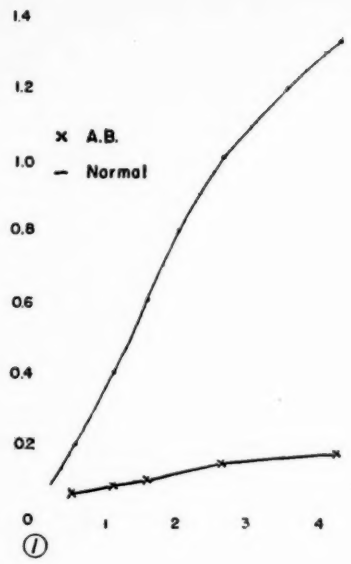
3. ACHROMATIC SENSITIVITY

Luminosity distribution in spectrum. Subject A. B. was tested to determine whether or not her luminosity curve was shifted toward the short-wave end of the spectrum. One half of the photometric field was illuminated with achromatic light to yield a constant luminance of 0.02 ml., while the other half received chromatic light from Wratten filters. The five filters that were used are listed in table 2. The measurements were made by varying the luminance of the chromatic light to match, for A. B., the achromatic. Similar determinations were made by a normal subject and, from the two sets of data, the luminosities of A. B. relative to the normal subject were computed. The results are given in table 2, the last column of which shows that the red, orange, and yellow-green are much less bright for A. B. than for the normal, while green

TABLE 2

LUMINOSITIES FOR A. B. RELATIVE TO A NORMAL SUBJECT WITH EACH OF FIVE CHROMATIC STIMULI

Wratten Filter No.	Wave Length at Max. Transmission	Normal Hue	Luminosity ratio A. B./Normal
70	700mμ	red	0.55
72	610	orange	0.116
73	570	yellow-green	0.623
74	530	green	1.76
76	440	blue	2.46



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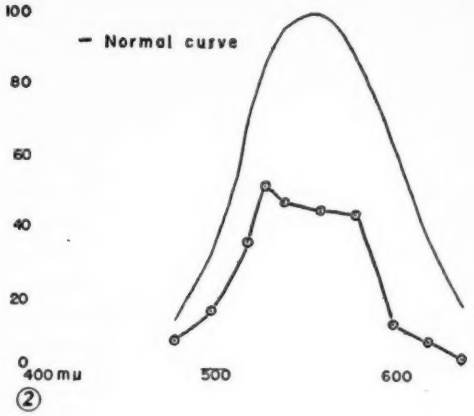

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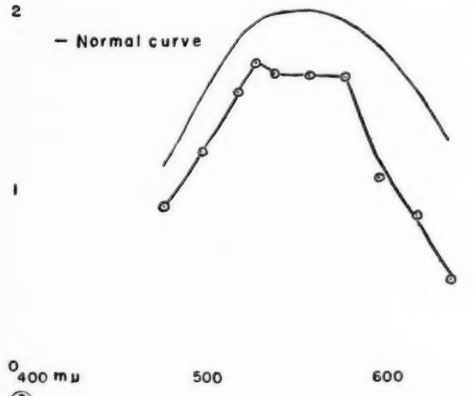

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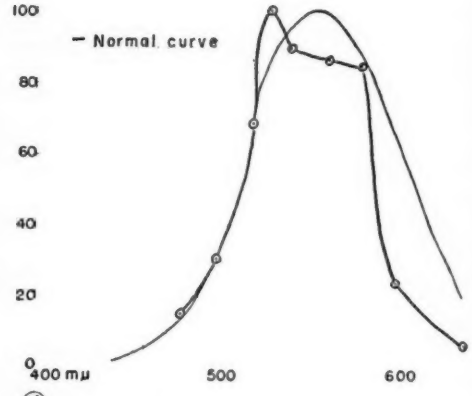

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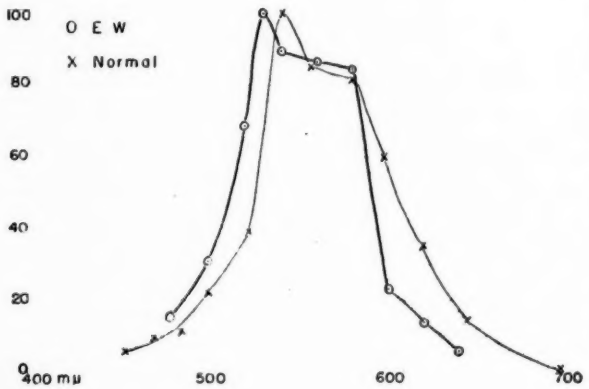

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Fig. 1 (Sloan and Newhall). Relation of acuity to luminance of background for A. B. and a normal subject. Luminance in log microlamberts = abscissae; acuity = ordinates.

Fig. 2. Luminosity curve of E. W. relative to that of a normal eye, as plotted in arithmetic units (table 3, col. 4). Luminosity = ordinates; wave length = abscissae.

Fig. 3. Luminosity curve of E. W. relative to that of a normal eye, as plotted in logarithmic units. Log luminosity = ordinates; wave length = abscissae.

Fig. 4. Luminosity curve of E. W. relative to that of a normal eye, as plotted with the maximum ordinates equated at 100 (table 3, col. 5). Luminosity = ordinates; wave length = abscissae.

Fig. 5. Luminosity curve of E. W. (maximum = 100) as compared with the luminosity curve of a normal dark-adapted fovea at 0.003 ml. Luminosity = ordinates; wave length = abscissae.

and blue are brighter than for the normal. These results indicate that A. B. probably has the typical shift in the luminosity curve with shortening of the red end and maximum luminosity in the green.

Similar determinations for E. W. showed no definite evidence of a shift in the luminosity curve. Therefore, more detailed observations were made with homogeneous radiation from 10 different regions of the spectrum between 480 mμ and 640 mμ. The subject was required to adjust the light, at each wave length, to match in brightness a standard achromatic stimulus maintained at a fixed luminance of 2.0 ml. Similar determinations were made by a normal subject. Table 3, column 2, shows at each wave length the computed luminosity relative to that for the normal eye. In the region between 480 mμ and 580 mμ, E. W.'s values approximate one half the normal; in the long-wave end of the spectrum, they approximate one fifth the normal.

To permit for E. W. computation of the luminosity curve of an equal-energy spectrum, the assumption was made that the luminosity curve of our normal subject did not differ significantly from that of the average normal.[40,41] The standard average data of Gibson and Tyndall are given in column 3. If we multiply these values by the corresponding values in column 2, the products (column 4) give for E. W. the luminosities throughout an equal-energy spectrum relative to those of a normal eye. The data in column 4 are charted in figure 2 and show, in comparison with the normal luminosity curve, a general reduction but no evidence of a true shift toward the short-wave end.

With the luminosities plotted in arithmetic units, as in figure 2, the deviations from normal are exaggerated in the mid-region and minified at the two ends of the spectrum. If the luminosities are plotted in logarithmic units, however, equal separations on the ordinate scale correspond to equal ratios of luminosity throughout the entire range of wave lengths. The data of figure 3 correspond to those of figure 2, except that the luminosities are shown in logarithmic scale. Figure 3 shows that there is a relatively constant decrease in luminosity of about 0.3 log unit in the region between 480 and 580 mμ and a greater decrease, about 0.7 log unit, beyond 580; but, again, there is no evidence of the characteristic shift in the curve as a whole toward the short-wave end of the spectrum. The decreased luminosity for all spectrum stimuli in

TABLE 3

COMPUTATION FOR E. W., OF THE LUMINOSITY CURVE OF AN EQUAL ENERGY SPECTRUM

(1) Wave Length mμ	(2) Luminosity Ratio E. W./Normal	(3) Luminosity,[40,41] Av. Normal	(4) Luminosity, E. W. rel. to Normal	(5) Luminosity, E. W. with Max. = 100
480	0.578	13.9	8.0	15.5
500	0.495	32.3	16.0	31.0
520	0.500	71.0	35.5	68.7
530	0.600	86.2	51.7	100
540	0.483	95.4	46.1	89.3
560	0.447	99.5	44.5	86.1
580	0.500	87.0	43.5	84.1
600	0.195	63.1	12.2	23.6
620	0.192	38.1	7.3	14.1
640	0.184	17.5	3.2	6.2

comparison with the luminosity of "achromatic" light, including all wave lengths of the visible spectrum, seems to contradict the principle that the luminosity of a mixture is equal to the sum of the luminosities of its components. The tendency to overestimate the brightness of chromatic stimuli is a difficulty peculiar to the equality-of-brightness method which might possibly account for this seeming contradiction.

The slight abnormality in the *shape* of curves are presented in figure 5, and their close resemblance suggests that E. W. has an abnormally functioning cone mechanism, the responses of which at ordinary levels of illuminance resemble those of the normal dark-adapted eye.

Field of vision for achromatic stimulus. The limits of the field of vision for a 1-degree white stimulus were determined on the Ferree Rand perimeter with the standard illuminance of 7 foot-candles.

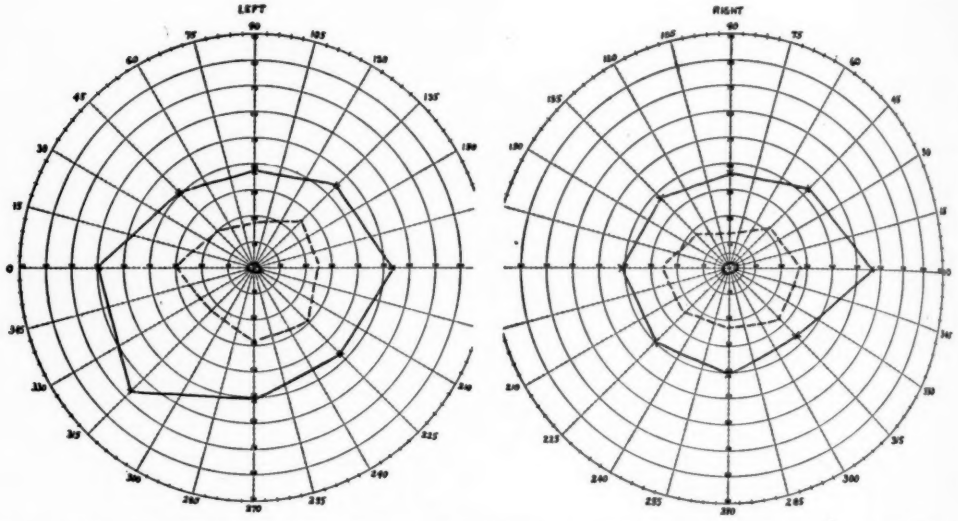

Fig. 6 (Sloan and Newhall). The visual fields of subject E. W. X = 1 degree white; broken line = 1 degree blue; solid line (at center) = 1 degree red. One-half degree red not recognized.

E. W.'s luminosity curve is shown more clearly in figure 4, where the relative luminosities at each wave length are expressed in units that make the maximum ordinate equal to 100, as in the normal luminosity curve. The shape of E. W.'s curve bears a close resemblance to the curve of the normal eye when the latter is based on photometric matches restricted to foveal cones in dark adaptation. A luminosity curve determined under such conditions—that is, with a normal dark-adapted eye and a 1-degree test field at a luminance of 0.002 ml.—has been taken from a previous publication by one of us[42] for comparison with E. W.'s curve. Both

The fields of A. B. were entirely normal in extent; those of E. W. showed a more or less concentric contraction of moderate amount, and are represented by the outermost contours in figure 6. The conditions under which the perimetric studies were made were such that the responses involved both rod and cone function. Therefore, further tests were made in which it was possible to differentiate between rod and cone response.

Rate of dark adaptation after previous light adaptation. The procedure for measuring the rate of dark adaptation included previous adaptation for three minutes to a

preëxposure field illuminated with achromatic light. The field was a horizontal oblong, 53 degrees by 62 degrees in extent. At the end of the three-minute period the light of the preëxposure field was turned off, and a series of threshold tests were made at intervals during dark adaptation. These thresholds were determined in the horizontal meridian of the nasal field at 15 degrees from the fixation point. The fixation point was an illuminated red target and the test area subtended a visual angle of 1 degree. The instrument and the method of making these determinations have been described elsewhere.[43,44]

Two luminances of preëxposure field were employed; namely, 1,100 and 100 ml. Adaptation of 1,100 ml. gives for the normal eye a curve of dark adaptation consisting of two separate sections. Kohlrausch[12,13] and, later on, Hecht,[45,46] explained these two portions of the curve as due, respectively, to cone and to rod adaptation. After adaptation to a preëxposure field of 100 ml., the curve of dark adaptation of the normal eye shows only the rod section, presumably because even in the early stages of dark adaptation the rod thresholds are lower than those of the cones.

The preliminary tests with the field luminance of 1,100 ml. showed that subject A. B. held the lids partially closed and kept up a steady and rapid blinking. No similar interference was encountered in her adapting to a luminance of 100 ml. Though the subject stated that the higher luminance produced neither pain nor particular discomfort, she was unable by voluntary effort to prevent the rapid blinking or partial closure of the lids. For the final tests, several drops of pontocaine were instilled into the eye shortly before the commencement of the three-minute period of light adaptation. During the three-minute exposure to the higher luminance the subject held up her upper lid with her finger. These expedients prevented partial closure and reduced the amount of blinking. In marked contrast to A. B., subject E. W. showed no noticeable tendency to avoid exposure to the high luminance of the preëxposure field.

The curves of dark adaptation for A. B., E. W., and the normal subject are shown in figures 7, 8, and 9. A. B.'s curve

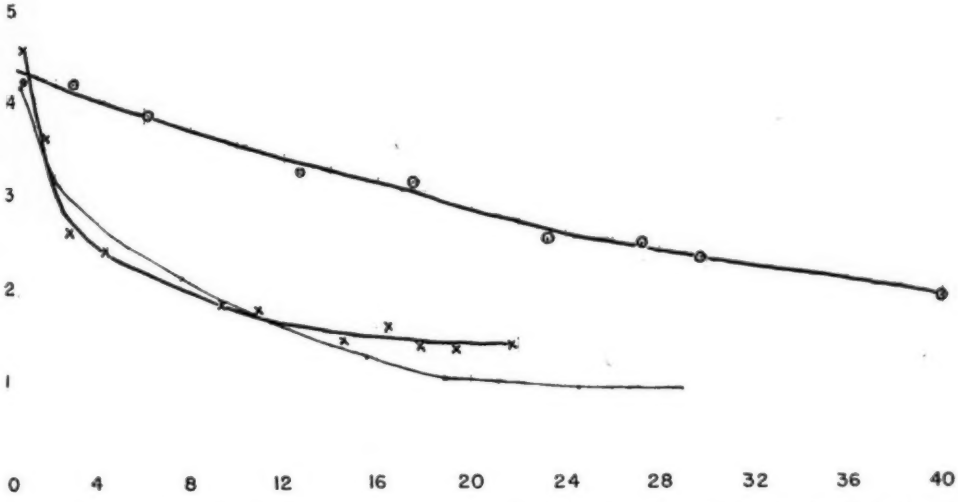

Fig. 7 (Sloan and Newhall). Rates of dark adaptation for A. B. (cross), E. W. (circle), and normal subject (solid), following preëxposure to 100 ml. Threshold—log μml. = ordinates; time in dark (minutes) = abscissae.

of dark adaptation after preëxposure to 100 ml. (fig. 7) did not differ significantly from the normal curve. E. W., however, exhibited an abnormally slow rate of decrease in threshold with dark adaptation. Figures 8 and 9 show the curves of dark adaptation after preëxposure to a luminance of 1,100 ml. The thresholds of

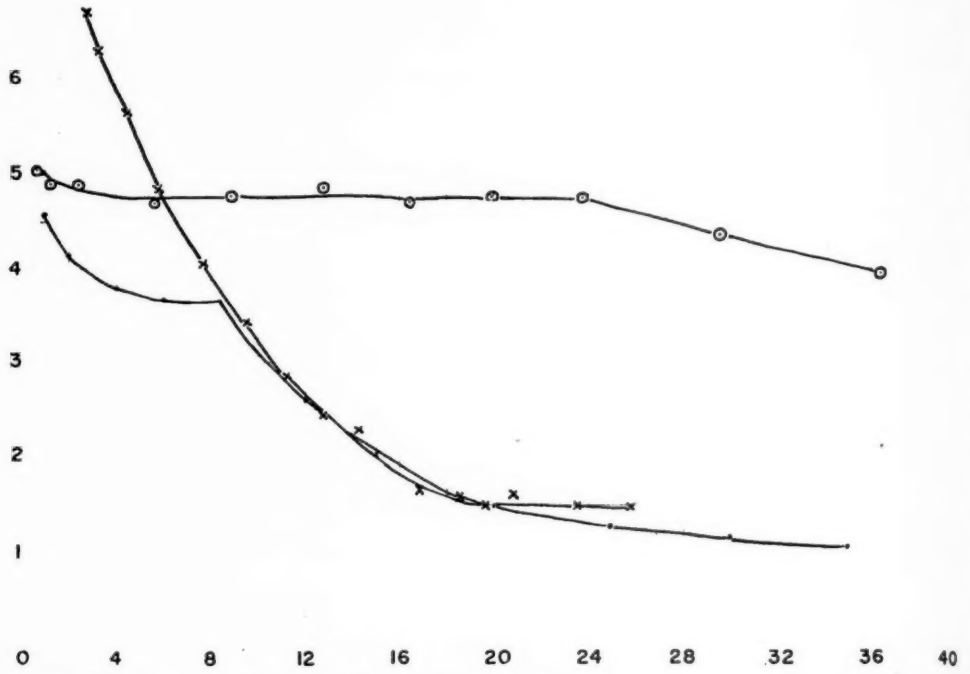

Fig. 8 (Sloan and Newhall). Rates of dark adaptation for A. B. (cross), E. W. (circle), and normal subject (solid), following preëxposure to 1,100 ml. Threshold—log μml. = ordinates; time in dark (minutes) = abscissae.

A. B. after about eight minutes of dark adaptation agreed closely with those of the normal eye. The shapes of the curves of A. B. and the normal subject indicate that during the first eight minutes of dark adaptation the thresholds of the normal eye are determined by the cones, whereas those of A. B. formed part of the curve of rod adaptation. After three minutes of dark adaptation A. B.'s threshold was about 2.5 log units above normal, or, in arithmetic units, about 300 times the normal value. The smoothness of the adaptation curves suggests that these abnormally high thresholds are consistent with normal rod sensitivity but are not found in the normal eye because during this stage the cones are able to respond to a much lower luminance. The adaptation curve of A. B. is, therefore, consistent with the view that rod function was entirely normal, whereas cone function was absent or greatly reduced.

As with preëxposure to 100 ml., E. W. showed an abnormally slow rate of dark adaptation, and differed markedly both from the normal curve and from that of subject A. B. At the beginning of dark adaptation E. W.'s threshold was close to the normal value, but there was almost no fall in the threshold until after about 24 minutes of dark adaptation. Then the threshold decreased very slowly but showed no indication of having reached

a final level, even after 80 minutes in the dark (cf. fig. 9). After 35 minutes of adaptation to darkness the threshold was about 3 logarithmic units above normal, after 80 minutes about 1 unit. The curves showing E. W.'s responses suggest that there must be some defect in the function of the rods as well as of the cones.

accept this slight evidence of a dual curve as proof of the existence of some cone function, the data indicate achromatic cone thresholds from 1.5 to 2 logarithmic units above normal, or, in arithmetic units, 70 to 100 times normal.

Measurement of the adaptation rate, it should be emphasized, gives data with

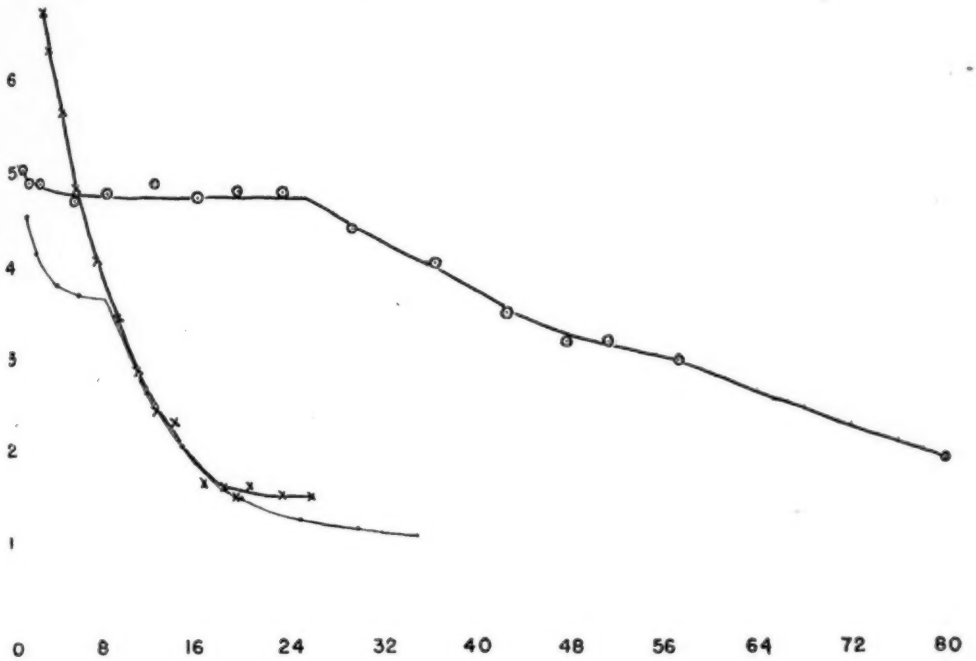

Fig. 9 (Sloan and Newhall). Rates of dark adaptation for A. B. (cross), E. W. (circle), and normal subject (solid), following preëxposure to 1,100 ml. (Same conditions as in figure 8 except longer dark adaptation for E. W.) Ordinates and abscissae as in figure 8.

In addition to the measurements following exposure to 100 ml. and 1,100 ml., A. B.'s dark adaptation rate was determined after preëxposure to 362 ml. The curve for A. B., in comparison with that of a normal subject, is shown in figure 10. The normal curve provides evidence that the cone thresholds are below those of the rods during the first four minutes of dark adaptation. Furthermore, the curve of A. B. showed a slight break at about four minutes, suggesting that prior to this time the thresholds may have been determined by cone response. If we can

regard to visual function for only a single region of the retina. Further tests were made, therefore, in which the thresholds after prolonged dark adaptation were determined at the fovea and at a series of locations in the horizontal meridian extending from the center of the retina to the extreme periphery. Since the rod threshold of the completely dark-adapted eye is well below that of the cones, the thresholds, except at the fovea, depend entirely upon rod response. Figure 11 shows the threshold curves for the two subjects, A. B. and E. W., in com-

parison with that of the average normal subject. The thresholds of A. B. presented no significant deviation from normal. The foveal threshold of the normal eye was higher than that of the immediately surrounding region, presumably because the dark-adapted foveal cones have a lower achromatic sensitivity than that of the similarly that the physiologic central scotoma, found normally in the dark-adapted eye, is also present in subjects with typical achromatopsia in whom no central scotoma is demonstrable during light adaptation. Best suggested that in order to explain these findings we must either assume that there are rods in the fovea that

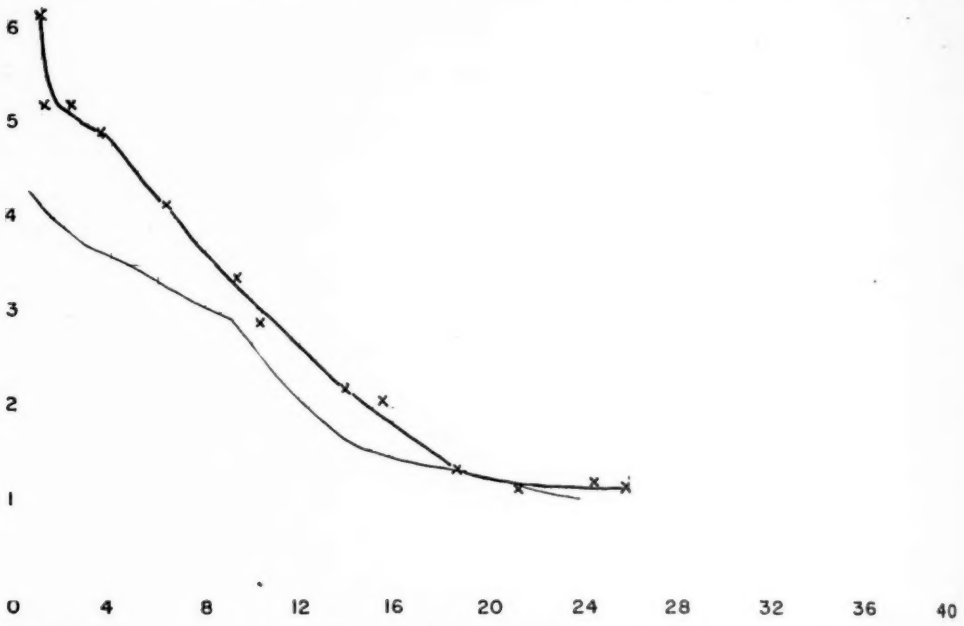

Fig. 10 (Sloan and Newhall). Rates of dark adaptation for A. B. (cross), and normal subject (solid), following preëxposure to 362 ml. Ordinates and abscissae as in figure 8.

rods in the parafoveal regions. The foveal threshold of A. B. was within normal limits, and, as in the normal eye, it was higher than the thresholds of the immediately surrounding regions. We must conclude, therefore, that in dark adaptation the achromatic sensitivity of A. B.'s fovea did not differ significantly from normal.*

Best[47] and others[10, 48, 49] have reported function differently from peripherally located rods, or that abnormally functioning cones are present that can mediate achromatic but not chromatic sensation. Hecht *et al.*[11] have suggested that the achromat may possess two kinds of rods.

The threshold curve of E. W. in figure 11 deviated markedly from the normal. In the peripheral field the curve was about 2 logarithmic units above the normal which indicates a 100-fold increase in the threshold there. In the more central regions the increase in threshold was less evident. Unfortunately, measurements of the foveal threshold were inadvertently omitted in this case.

This last test, like the preceding one,

* The pericentral fixation target used in making determinations at the fovea has been described elsewhere.[48] It should be mentioned here that measurements of foveal threshold are subject to considerable error and show variability even in normal subjects because of the difficulty of maintaining fixation during the measurements.

suggests that rod function was normal in the case of A. B. and definitely defective in the case of E. W.

4. OPHTHALMOSCOPIC EXAMINATION

Careful ophthalmoscopic examination of both subjects revealed normal fundi, normal optic nerves, and no evidence of any pathologic changes that might account for the visual abnormalities. The fact that abnormalities had apparently been present from birth in both subjects is further evidence that the effect is congenital and not due to any ocular pathology.

5. COLOR BLINDNESS IN FAMILY HISTORY

A. B.'s maternal grandfather was reported to have defective chromatic vision. Details as to the type of his defect were not obtainable. A. B.'s father, mother, and brother are known to have normal color vision. This pedigree is consistent with a simple recessive rather than with a sex-linked heredity. E. W.'s father, brother, and maternal uncle were reported to have had abnormal chromatic vision. No further details as to the type of the defect were obtainable. Baetjer[50] published the pedigree of this subject in 1922, and showed that E. W.'s chromatic blindness could be explained on the basis of sex-linked heredity. The possibility of recessive heredity, however, cannot be definitely ruled out.

The studies of the third subject of this report, J. W., were less complete than those of A. B. and E. W. The results may be summarized as follows: There was incomplete loss of chromatic vision for red, yellow, green, and blue. The corrected visual acuity of each eye was 20/100. Very fine nystagmoid movements of the eyes were noted when fixation was attempted. Under intense light stimulation, the subject showed a tendency to blink and close his lids partially. These manifestations of photophobia were, however, much less marked than those of subject A. B. under the same external conditions. Unlike A. B., this subject could prevent the manifestations by voluntary effort. The visual anomalies apparently had been

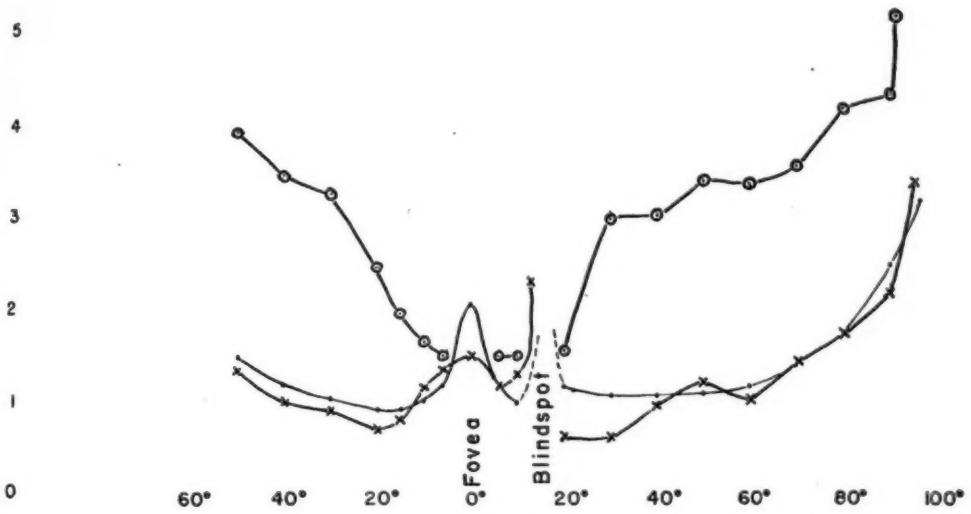

Fig. 11 (Sloan and Newhall). Achromatic thresholds in full dark adaptation for A. B. (cross), E. W. (circle), and the average normal (solid), as determined at selected points in the horizontal meridian. Threshold—log μml. = ordinates; nasal to left, temporal to right of 0 degree = abscissae.

present from birth. No other members of the family were known to have similar visual defects.

Photometric measurements with red and blue lights showed no significant deviation from normal. This result suggests that the typical shift in the luminosity curve was not present.

Curves showing the rate of dark adaptation after previous light adaptation to luminances of 1,100 ml. and 382 ml., respectively, proved to be essentially the same as those of A. B. (figs. 8 and 9). His final thresholds in the horizontal meridian were in close agreement with the normal. Thus, for J. W. there was evidence of marked increase in the cone thresholds and of normal rod thresholds.

Comment

Absent or defective cone function explains cases of typical achromatopsia fairly well. There are, however, several experimental facts which are not entirely in accord with the assumption of absence of cones. *First,* Larsen[38] found that cones *were* present in one such case and showed abnormal structure in the central regions but not in the periphery. This finding suggests that achromatopsia is due to a defect in cone function rather than to complete absence of cones. *Second,* measurements of the adaptation rate of our subject A. B. at 15 degrees in the nasal field gave results suggesting that cone function might be deficient but not entirely absent; for after adaptation to a moderate level of luminance, the curve of dark adaptation evinced two sections, a cone section with thresholds well above normal limits, and a rod section with thresholds in the normal range. *Third,* the threshold gradient curve of A. B., as determined in the horizontal meridian under full dark adaptation, revealed a normal course with higher thresholds at the fovea than in the paracentral and peripheral regions.

Best[47] and others[10, 48, 49] reported subjects with cases of typical achromatopsia in whom the normal physiologic central scotoma could be demonstrated in the fully dark-adapted eye, although no scotoma was demonstrated in light adaptation. The increased foveal threshold could be accounted for by assuming that rods were substituted for cones at the fovea; then the higher threshold in dark adaptation might be due to a lesser density of rods in the fovea than in the surrounding regions. An alternative explanation would be on the assumption that the fovea contained cones which functioned normally only when dark adapted.

The cases of achromatopsia reported in the literature, which we have classified as atypical, include subjects with incomplete loss of chromatic vision for one or more colors, subjects without nystagmus, photophobia or reduced foveal acuity, and those lacking the typical shift in the luminosity curve. The entire group of atypical features were not, as a rule, reported present, so that as described in the literature these individuals do not form a homogeneous group. For that reason alone, it would be difficult to provide any completely satisfactory explanation of the atypical forms of achromatopsia. Our subject J. W. was apparently atypical only because chromatic vision was not completely lacking and there was no shift in the luminosity function. E. W., on the contrary, exhibited all of the atypical characteristics listed above. She also showed an abnormally slow rate of dark adaptation, and, furthermore, the final threshold was markedly above the normal for all retinal regions from center to periphery. This retardation of dark adaptation and elevation of the final threshold level suggest some defect in the rod mechanism in addition to the defect in the cone mechanism.

It seems clear that the anomalies in visual perception found in atypical achro-

matopsia cannot be explained adequately on the simple assumption of a defective cone mechanism. Until there is a well-established basis for the facts of normal color perception there is little hope of really explaining the various forms of partially defective color vision. Polyak,[51] in a recent monograph on the anatomy and histology of the retina, points out that there is no anatomic evidence for the existence of different types of cones to mediate the several primary color sensations. He suggests that the several varieties of retinal bipolar cells could provide the primary chromatic analysers needed to explain color vision. The cause of the various types of color blindness, he writes, is probably a defect in the biochemical constitution of one or more of the varieties of bipolar cells, rather than their actual absence. "The defect may be due," he suggests, "merely to an incomplete differentiation of the bipolar cells into clear-cut types, each entrusted chiefly with the perception of a portion of the spectrum."

The little-known theory of Adams[52] not merely posits the importance of the bipolar cells but goes much further in providing an explicit schema of their conceived role in accounting for various forms of achromatopsia and partial color blindness. His first form is the typical one to which our subject A. B. conformed so closely. His second form is the atypical variety characterized by normal acuity, no shift in luminosity, and night blindness. Our subject E. W., with her abnormally high thresholds in darkness, evinced all of these characteristics. It would seem that this neglected theory merits serious review at a time when so many indicators are pointing to the importance of mechanisms central to the rods and cones.

Summary

Achromatopsia in its typical form shows the following characteristics:

(1) There is either complete absence of chromatic vision or markedly defective chromatic vision for blue and yellow as well as for red and green. (2) There is reduced foveal acuity. (3) The luminosity curve of the spectrum at high intensities resembles the normal scotopic luminosity curve. (4) Photophobic symptoms and nystagmoid eye movements are usual. (5) The defect is inherited as a simple recessive characteristic.

Detailed studies were made of two subjects, one of whom conformed closely to the typical form of achromatopsia while the other was anomalous in various respects. The results are as follows:

(1) Both subjects, tested with the Ishihara charts, showed greater defects than those characteristic of simple red-green color blindness.

(2) In photometric matches between achromatic and chromatic stimuli, and in perimetric tests with red and blue Heidelberg papers, the first subject evinced complete absence of chromatic vision while the second showed very weak chromatic responses for red, yellow, and blue.

(3) The first subject gave evidence of a typical scotopic luminosity curve. The second showed decreased luminosity throughout the spectrum, and a slight shift in the region of maximum luminosity. There was a striking difference in the rates of dark adaptation of the two subjects. The first evinced normal rod response and absent or markedly deficient cone response; whereas the second showed an abnormally slow rate of dark adaptation, pointing to defective functioning of both rods and cones.

(4) The first subject evinced defensive blinking, marked nystagmus, and reduced foveal acuity. The second was normal in all of these respects. A central scotoma was not demonstrable in either subject.

(5) Ophthalmoscopic examinations were entirely negative, indicating that the

visual defects in both cases were congenital rather than pathologic. The pedigree of the first subject is consistent with simple recessive heredity, while the heredity of the second could be sex-linked.

Less extensive tests were made of a third subject who revealed only a partial loss of chromatic vision and no shift in the luminosity curve. In all other respects the findings from this third subject were those of typical achromatopsia.

Our results from the subject with typical achromatopsia can be attributed to complete or almost complete absence of function of some essential component in the mechanisms primarily responsible for photopic vision. No more specific physiologic basis for this defect can be definitely assigned at present.

The various forms of partially defective chromatic vision such as (a) the type of defect exemplified by our second subject with incomplete loss of chromatic vision for blue and yellow as well as for red and green, (b) complete red-green color blindness with normal vision for blue and yellow, and (c) incomplete red-green color blindness, likewise cannot be adequately explained without further knowledge of the physiology of the retina and possibly of the entire visual pathway.

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FIVE HUNDRED MELANOMAS OF THE CHOROID AND CILIARY BODY FOLLOWED FIVE YEARS OR LONGER*

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The American Registry of Ophthalmic Pathology maintained at the Army Medical Museum under the auspices of the American Academy of Ophthalmology and Otolaryngology now contains 1,600 cases of uveal melanoma. This accumulation consists of contributions of single specimens sent in for examination and of representative sections from specimens in the larger collections throughout the country. Acknowledgment has been made in a previous paper[1] to those persons who have been most active in amassing this material. At the present time it is our desire to thank the contributors for their splendid coöperation in our follow-up system, which has made possible the studies on prognosis.

Of the aforementioned 1,600 cases, 500 cases of malignant melanoma of the choroid and ciliary body, which form the basis for this paper, have been followed five years or more. That is, the patients are known either to have lived at least five years after the eye had been enucleated and recurrence or metastasis did not take place, or to have died as a result of metastasis. Cases in which the eye was enucleated later than 1936 are not included in this study. Among the 261 patients classified as "living," 22 died as a result of unrelated causes between 5 and 16 years following enucleation of the eye, and 13 died as a result of unknown causes between 5 and 24 years postoperatively. Among the patients who died as a result of metastases the shortest postoperative period was 2 days, the longest, 17 years, and the average, 37 months. The patient who lived 17 years after the eye had been enucleated had a melanoma of the skin of two months' duration excised one year before death. At the time of death multiple melanomas of the skin were present as well as metastases to the liver and other organs of the body. As to patients who live many years following enucleation of an eye affected by malignant intraocular melanoma and who die as the result of a malignant tumor situated elsewhere in the body, there is always a question as to whether the tumor eventuated from actual metatases from the original melanoma or was an unrelated primary growth which developed later. Tumors arising only in the choroid or in the ciliary body and involving the choroid are included in the tables on classification and prognosis which accompany this report. Thirty-two cases of tumor of the iris observed five years or longer were set aside for separate study because of several factors that appeared to make them unsuitable for inclusion in this group. Many of them did not fall into the six histologic classifications on which our prognostic studies have been based. Frequently tumors of the iris closely resemble nevi of the skin. Their location also is a disturbing factor in as much as these tumors, because of their visibility through the cornea, are usually recognized much earlier than those of the ciliary body or choroid. Only three deaths have occurred from tumors obviously arising in the iris. Three patients on whom iridectomy only was performed are alive seven years after operation.

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Among the 1,600 cases of malignant intraocular melanoma contained in the Registry, eight diagnoses were made in Negroes none of whom has been observed for a period as long as five years. This rarity of melanomas occurring among the Negro race is the most striking feature of our data on incidence. There is a slight preponderance of males over females and of deaths among males over deaths among females (tables 1 and 2).

TABLE 1

SEX DISTRIBUTION

Male	799
Female	751
Unknown	50
Total	1,600

The distribution of age (tables 3 and 4) is of considerably greater interest than the incidence in sex. The age of the youngest patient suffering from typical malignant melanoma of the choroid reported in the Registry is 11 years, that of the oldest is 91 years. The age of the youngest patient

TABLE 2

OUTCOME ACCORDING TO SEX

Sex	Living	Dead	Total	Percent Dead
Male	121	131	252	52
Female	139	106	245	43
Unknown	1	2	3	66
Total	261	239	500	48

reported in the group of 500 is 17 years, that of the oldest 88 years. In actual numbers patients in the sixth decade of life suffering from this disease seem to outnumber those in any other decade; in proportion to population[2] the incidence appears highest in the seventh and eighth. The highest death rate among the cases observed occurs in patients in the eighth and ninth decades of life. One patient whose eye was enucleated at the age of 19 years, died four years later as a result of general metastases. This is the youngest patient in our series to die as a result of metastases from malignant intraocular tumor.

TABLE 3

COMPARISON OF THE PERCENTAGE DISTRIBUTION BY AGE GROUPS OF 1,418 CASES OF MALIGNANT MELANOMA WITH PERCENTAGE DISTRIBUTION OF THE POPULATION BY AGE GROUPS*

Age Group years	No. of Cases	Percentage Distribution of Cases	Percentage Distribution of Population
0– 9	2	0.14	20.8
10–19	13	0.92	17.9
20–29	71	5.01	17.2
30–39	189	13.33	15.4
40–49	296	20.87	11.8
50–59	375	26.44	8.4
60–69	328	23.13	5.1
70–79	130	9.17	2.3
80–89	13	0.92	0.6
90–99	1	0.07	0.5
Total	1,418	100.00	100.0

In 182 cases the age was unknown.
* 1920 Census, white males and females.

The following descriptions are a brief review of the bases for cell and fiber classifications published in previous papers:[1, 3, 4]

TABLE 4

OUTCOME ACCORDING TO AGE

Age Group years	Living	Dead	Total	Percent Dead
10–19	3	1	4	25
20–29	9	10	19	53
30–39	41	19	60	32
40–49	61	39	100	39
50–59	57	68	125	54
60–69	57	45	102	44
70–79	12	18	30	60
80–89	1	2	3	67
Unknown	20	37	57	65
Total	261	239	500	48

Spindle A (fig. 1): Spindle-shaped cell with a narrow oval nucleus and indistinguishable or ill-defined nucleolus. Frequently there is seen a line or fold ex-

tending through the center of the nucleus in its long axis.

Spindle B (fig. 2): Spindle-shaped cell in which the nucleus is oval but generally more robust than the spindle-A nucleus.

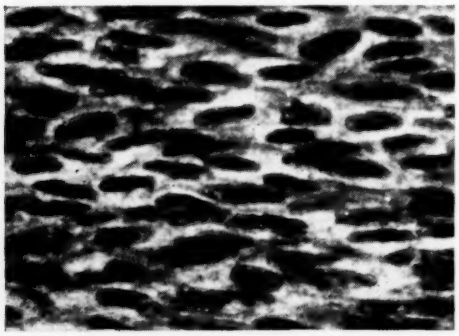

Fig. 1 (Callender *et al.*). Spindle cell, subtype A. Hematoxylin and eosin. ×705. A.M.M. Negative No. 72084.*

There is a prominent nucleolus. *Fascicular* (fig. 3): The term describes the palisaded arrangement of the cells rather than their morphology. The cell resembles the spindle-B type. *Epithelioid* (fig. 4): Round or polygonal cells, usually rather

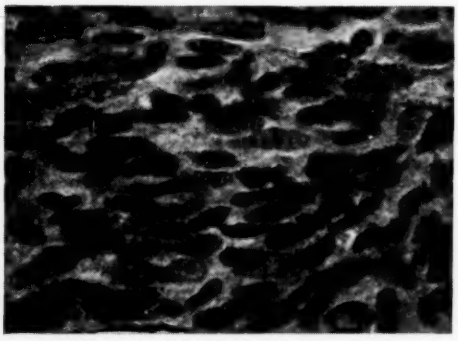

Fig. 2 (Callender *et al.*). Spindle cell, subtype B. Hematoxylin and eosin. ×705. A.M.M. Negative No. 72081.

large but with considerable variation in size and shape. The nucleus is large, round, and nucleolated. Multinucleated forms are frequently seen. The cytoplasm is abundant, usually homogeneous, and markedly acidophilic. *Mixed:* A combination of two or more types, except spindle-A and -B tumors, which are all classed as spindle-B, and spindle-B and fascicular tumors, which are classed as fascicular. *Necrotic:* Too necrotic for accurate classification.

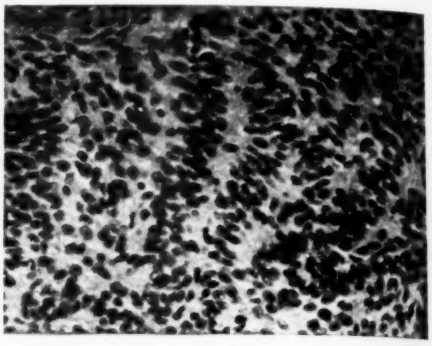

Fig. 3 (Callender *et al.*). Fascicular type. Hematoxylin and eosin. ×230. A.M.M. Negative No. 72082.

The fiber classification is based on the relative content of argyrophil fibers as demonstrated by the Wilder reticulum stain.[5]

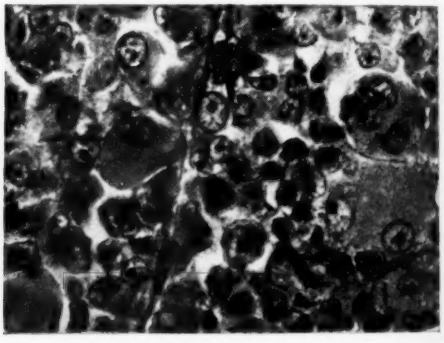

Fig. 4 (Callender *et al.*). Epithelioid-cell type. Hematoxylin and eosin. ×500. A.M.M. Negative No. 72085.

Heavy (fig. 5): Tumors having in all areas fibers forming a network about individual tumor cells. *Marked:* Tumors having a definite preponderance of areas

* All illustrations are from Army Medical Museum files.

containing fibers, fiber content more than 50 percent. *Medium* (fig. 6): Tumors having areas with and without fibers approximately equal, fiber content about 50 percent. *Light:* Tumors having a definite preponderance of fiberless areas, fiber content less than 50 percent. *Absent* (fig. 7): Tumors having no fibers or fibers only in the interlobular stroma.

As nearly as possible the sections in each case have been taken through the approximate center of the tumor and to include that part of the uveal tract from which it arises.

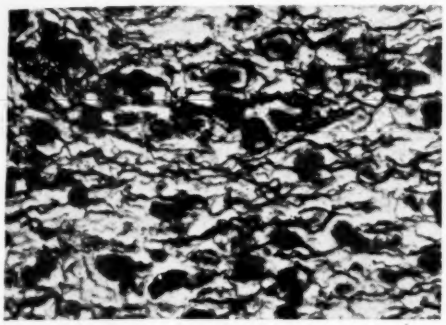

Fig. 5 (Callender *et al.*). Heavy fiber content. Wilder reticulum stain. ×400. A.M.M. Negative No. 63412.

Since the last tables published in 1939, the number of patients followed five years or more (tables 5, 6, 7) has nearly doubled. The percentage of deaths as a result of malignant intraocular melanoma, however, remains approximately the same: 49 percent in 1939, 48 percent of the present group. Although variations in percentages of deaths have occurred in the groups comprising the cell and fiber classifications, the relative malignancy of these types of tumors remains the same. With the doubling of cases, however, there have been two deaths in the spindle-A group. One of the tumors was devoid of fibers and the other contained a marked number of them. Two deaths have also occurred in the heavily fibered group; both of the tumors were of the mixed-cell type. Five of the patients are living in whose cases the tumors contained no argyrophil fibers and from whom the tumors were removed more than five

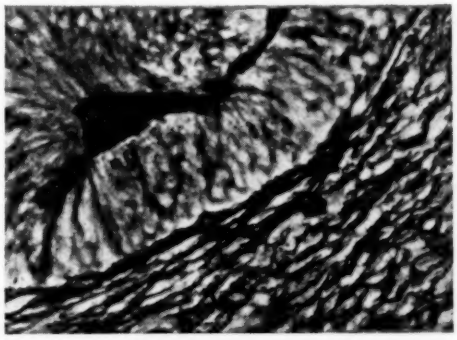

Fig. 6 (Callender *et al.*). Fibered and unfibered areas. Wilder reticulum stain. ×400. A.M.M. Negative No. 66363.

years ago. The percentage of deaths in this group, therefore, is reduced from 100 to 80 percent. In the spindle-B group there has been an increase in the percentage of deaths, from 21 to 25 percent. In the fascicular group, where the number of cases increased from 4 to 13 cases,

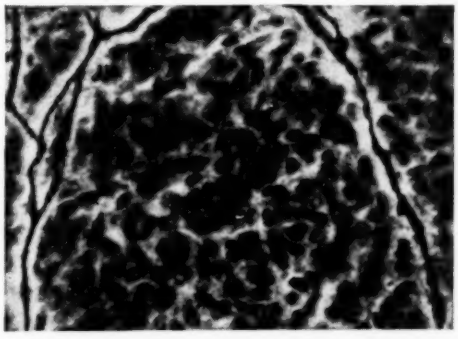

Fig. 7 (Callender *et al.*). Fibers absent except in interlobular stroma. Wilder reticulum stain. ×400. A.M.M. Negative No. 66360.

the percentage of deaths has dropped from 80 to 38, which demonstrates the potential inaccuracy of conclusions based on a few cases. In the epithelioid group, however, where the number of cases has

increased from 5 to 14, the percentage of deaths has decreased from 80 to 71, which is the highest death rate occurring in any group. As in the spindle-B group, the variation in percentage of deaths in the mixed group remains slight even

TABLE 5

OUTCOME ACCORDING TO CELL TYPE

	Living	Dead	Total	Percent Dead
Spindle A	33	2	35	6
Spindle B	88	29	117	25
Fascicular	8	5	13	38
Necrotic	23	22	45	49
Mixed	105	171	276	62
Epithelioid	4	10	14	71
Total	261	239	500	48

though the number of cases has doubled in the later table: 65 percent in the former table and 62 percent at the present time.

Worthy of consideration is a group of 200 patients who have been followed 10 years or more, in which group the percentage of deaths has risen from 48 to 66. Of these there are 41 patients in the group of 239 who died as a result of metastases, six of whom lived more than 10 years. It is obvious that, although the average duration of life postoperatively is 46 months, even in the 10-year group, patients who live five years after enucleation are still not completely out of danger from metastasis.

The 500 cases followed have also been classified according to their pigment content (table 8). There appears to be an increase of malignancy with an increase of pigmentation, but it is so slight that a greater number of cases must be studied before conclusions based on this classification are drawn.

It is not always possible, particularly where sections alone are contributed, to determine whether or not extrabulbar extension has occurred. It has been demonstrated in 81 patients whose cases were followed; of these 54, or 67 percent, have died as a result of metastases. Of these 81 patients, seven show optic-nerve involvement, apparently beyond the point of excision.

TABLE 6

OUTCOME ACCORDING TO ARGYROPHIL-FIBER CONTENT

	Living	Dead	Total	Percent Dead
Heavy	17	2	19	10
Marked	58	32	90	36
Medium	80	63	143	44
Light	101	122	223	55
Absent	5	20	25	80
Total	261	239	500	48

SUMMARY

There are 1,600 cases of malignant intraocular melanoma on record in the American Registry of Ophthalmic Pathology. Only eight of these are in Ne-

TABLE 7

CLASSIFICATION BY CELL TYPE AND FIBER CONTENT

Cell Type	Fiber Content									
	Heavy		Marked		Medium		Light		Absent	
	L	D	L	D	L	D	L	D	L	D
Spindle A	1	1	9		9		6	1	8	
Spindle B		2	34	16	32	4	15	7	7	
Fascicular		1	6	4	1		1			
Necrotic		4	7	11	11	5	4	2	1	
Mixed	4	11	44	87	25	49	31	22	1	2
Epithelioid		1	1	4	2	5	1			

groes. There is a slight preponderance of males over females. The tumor is one peculiar to adult life and appears but rarely in the earlier decades. Five hundred patients suffering from melanoma of the choroid and ciliary body have been followed five years or longer, or have died as a result of metastases before the expiration of that time. The tumors in this group have been classified by cell type and fiber content, and the following corollaries have been drawn: The pure spindle-cell types, particularly subtype A, are of a relatively low degree of malignancy, and an increased degree of malignancy is associated with the presence of epithelioid cells; a low degree of malignancy is usually associated with a heavy fiber content.

TABLE 8

OUTCOME ACCORDING TO PIGMENT CONTENT

	Living	Dead	Total	Percent Dead
Light	93	48	141	34
Medium	114	121	235	51
Marked	50	61	111	55
Heavy	4	9	13	69
Total	261	239	500	48

Although there appears to be an increased lethality with increased pigment content, it is felt that there is not enough variation in death percentages to justify conclusions until a larger number of followed cases has been accumulated. The total number of metastatic deaths is 239 among the cases followed five years or longer, or 48 percent. This percentage increased to 66 in 200 of these cases followed 10 years or longer.

The writers are particularly grateful to Major J. H. McNinch, Medical Corps, U.S.A., for his Chi square evaluations of our statistical studies and to Mr. Lawrence P. Ambrogi for technical assistance.

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CHRONIC OPHTHALMOPLEGIA EXTERNA*

Classification of causes. Report of a case

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Ocular paralysis may involve several eye muscles simultaneously, or may select individual muscles. *Ophthalmoplegia interna* designates paralysis of the intrinsic ocular muscles only (that is, the pupillary sphincter and ciliary muscles). *Ophthalmoplegia externa* refers to paralysis of the extrinsic muscles of the eye, while *ophthalmoplegia totalis* comprises paralysis of all the extrinsic and intrinsic eye muscles.[1] In onset and duration, ophthalmoplegia may be acute, subacute, or chronic, depending upon the etiologic factors. The following outline includes almost all of the known possible causes of chronic ophthalmoplegia externa:

I. Primary, or idiopathic, or nuclear ophthalmoplegia externa.
   A. Congenital familial ophthalmoplegia
   B. Chronic progressive ophthalmoplegia

II. Ophthalmoplegia secondary to:
   A. Trauma—particularly skull fractures
   B. Inflammation
      1. Meningitis
      2. Sinusitis
      3. Encephalitides
         a. Epidemic encephalitis
         b. Encephalitis complicating acute infectious diseases such as measles, smallpox, influenza, scarlet fever, varicella
      4. Polyneuritis, such as may occur in the course of diphtheria, rheumatic fever, pneumonia, diabetes, chronic alcoholism, pregnancy, and other diseases
      5. Inflammation of the orbit or periorbital tissues
      6. Botulism
      7. Syphilis, especially tabes dorsalis and meningovascular syphilis
      8. Herpes zoster ophthalmicus
   C. Increased intracranial pressure
      1. Neoplasms
      2. Hydrocephalus
      3. Brain abscess
   D. Circulatory changes
      1. Cerebral hemorrhage, thrombosis, or embolism
      2. Cavernous-sinus thrombosis
      3. Intracranial aneurysm
   E. Poisonings
      1. Alcohol poisoning—including acute polioencephalitis hemorrhagica superior of Wernicke
      2. Lead poisoning
   F. Miscellaneous causes
      1. Multiple sclerosis
      2. Myasthenia gravis
      3. Exophthalmic ophthalmoplegia
      4. Ophthalmoplegic migraine
      5. Periodic oculomotor palsy

Primary chronic ophthalmoplegias

Primary, or idiopathic, or nuclear chronic progressive ophthalmoplegia externa may be congenital, or may develop in later life, and may be familial. In 20 of 38 cases mentioned by McMullen and Hine,[2] the age of onset was under 20

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years, in two cases between 21 and 30 years, and in six cases over 30 years. In the cases that are not congenital, the onset is gradual and usually starts with ptosis, as a rule developing in childhood. There are no accompanying systemic disturbances or neurologic signs. The ptosis may be unilateral or bilateral. As in the case presented here, one eye may be affected several years before the other. The progress of the disease is very slow and may spread over three to four decades,[3] with the possibility that it may come to a standstill at any time. The ultimate result, if the disease progresses, is a complete bilateral external ophthalmoplegia. Diplopia may be present in the earlier stages[4, 5, 6] if there is asymmetrical involvement of the ocular muscles, and, occasionally, intermittent headaches may be present.[2] In a discussion of the congenital familial type of external ophthalmoplegia, Helfand[7] reviews the theories of origin of the congenital forms, which may be listed as follows:

1. Circulatory theory, which postulates that the ophthalmoplegia is the result of hemorrhages in the sheaths of the orbital nerves, or hemorrhages in the brain.
2. Neurogenic theory, which attributes the disease to a slow degeneration of the nuclei of the oculomotor nerves.
3. Myopathic theory, the proponents of which claim that the paralysis is due to a primary atrophy of the ocular muscles with secondary retrograde degenerative changes in the oculomotor nuclei.
4. Traumatic theory, attributing the paralysis to birth injuries to the efferent nerves, again with retrograde nuclear degenerative changes.

Similar theories have been postulated to explain the noncongenital type of chronic progressive external ophthalmoplegia. There is some dispute in the literature[5] as to whether the congenital ophthalmoplegias are the same in pathogenesis and progress as the noncongenital forms; however, in a short but comprehensive review of the subject McMullen and Hines[2] express the belief that there is very little difference between many cases of congenital ophthalmoplegia externa and some of those developing later in life. The most widespread theory is that the disease, in its "idiopathic" form, is probably due to an abiotrophy or a hypoplasia of the nuclei of the nerves to the eye muscles. Oppenheimer (quoted by McMullen and Hines) stated that the majority of cases of chronic ophthalmoplegia represent prodromata or local manifestations of central-nervous-system disorders, such as tabes dorsalis, general paresis, subacute combined sclerosis, multiple sclerosis, progressive bulbar paralysis, progressive muscular atrophy, or chronic anterior poliomyelitis. However, there is a group of cases in which the external ophthalmoplegia, although chronic and progressive in character, is not associated with any other evidence of neurologic disease. It is important in prognosis, therefore, to determine, if possible, to which particular group a patient belongs; however, aside from the prognostic implications, the differentiation, clinically, is purely academic, since in both forms (that is, with or without the later development of central-nervous-system disease) the ophthalmoplegia is external, is chronic, and is progressive.

Secondary chronic ophthalmoplegias

Trauma. The influence of trauma in producing ophthalmoplegia is obvious, since disruption of the functional continuity of the efferent nerves will result in a corresponding ocular paralysis. Fractures of the base of the skull or through the middle cranial fossa are particularly likely to produce ocular paralysis.

Inflammations. Systemic infectious processes may produce ophthalmoplegia by virtue of a complicating peripheral neuritis of the efferent oculomotor nerves (such as may occur in diphtheria, rheumatic fever, pneumonia, diabetic polyneuritis) or by involvement of the nuclei of the oculomotor nerves secondary to encephalitis complicating acute infectious diseases, such as measles, smallpox, influenza, scarlet fever, varicella, poliomyelitis.[8] Meningitis may result in ophthalmoplegia by involvement by contiguity of the motor nerves of the eye. A similar mechanism is responsible for abducens paralysis occurring in osteomyelitis of the petrous pyramid. Periostitis or fibrositis of the orbital tissues about the sphenoidal fissure may produce peripheral palsies of the sixth, fourth, and third cranial nerves, and the first and second divisions of the fifth cranial nerve, without involving the optic nerve or the ophthalmic blood vessels.[9] A similar mechanism resulting, however, from mechanical rather than inflammatory constriction of the sphenoid fissure, may produce ophthalmoplegias in osteitis deformans or other diseases of the cranial bones.[10] In botulism, oculomotor disturbances such as ptosis, mydriasis, loss of light reflex, or diplopia may be the initial manifestations and are typically due to third-nerve involvement.

Tabes or general paresis may be preceded by a progressive chronic ophthalmoplegia.[11] Carmody reports 150 cases of herpes zoster ophthalmicus in which 24 patients had paresis of the ocular muscles, the majority having third-nerve involvement.[12]

Increased intracranial pressure. Tumors may cause ophthalmoplegia by pressure either upon the nuclei of the oculomotor nerves, or upon the nerves themselves as they course within the cranium. Internal hydrocephalus, and neurofibromata involving the nerve sheaths, may produce similar manifestations.

Circulatory changes. Diseases of the vessels supplying the nuclei of the nerves may result in degenerative changes in the nuclei, with consequent ophthalmoplegia.

Intracranial aneurysm produces its effects primarily by pressure upon the nerve.

Poisonings. Lead and alcohol poisonings may produce a peripheral neuritis of the oculomotor nerves, and thus induce ophthalmoplegia. Acute polioencephalitis hemorrhagica superior (Wernicke's disease), whether it be due to alcoholism, avitaminosis, or infection, may cause bilateral ophthalmoplegia externa of acute onset.

Miscellaneous. Chronic progressive ophthalmoplegia may sometimes be a precursor of, or a development in the course of, multiple sclerosis, amyotrophic lateral sclerosis, or progressive nuclear degeneration. Myasthenia gravis may make itself manifest in only the ocular muscles for many years. A large number of cases of myasthenia gravis present ptosis as the first symptom. The internal ocular muscles are spared in this disease, but the external muscles are involved in 30 percent of cases.[13] The diagnostic test for this disease is, of course, the use of prostigmine. It is not generally appreciated that the ocular paresis accompanying myasthenia may become a total and permanent paralysis.

Exophthalmic ophthalmoplegia is an interesting entity, differing from exophthalmic goiter in that males are more frequently affected, and the symptoms of hyperthyroidism are mild, and may even be absent.[14] The ophthalmoplegia and

exophthalmos may arise spontaneously or may develop days or years after thyroidectomy, in which latter case, the basal metabolic rate may be normal or subnormal. One or both eyes may be affected, the exophthalmos being more pronounced than that seen in Graves's disease and associated with chemosis, edema of the lids, and sometimes with papilledema.[15] The etiology of this disease is unknown, but the ophthalmoplegia may be attributable to the mechanical effect of the intraorbital pressure on the ocular muscles.[13] Ophthalmoplegic migraine and periodic oculomotor palsy are ill-defined dysfunctions whose manifestations are obvious from their names.

Case report

H. S., a married white male laborer, aged 45 years, was admitted to the U. S. Marine Hospital, Detroit, on September 18, 1941, complaining of ptosis of the left eyelid of 30 years' duration, and of ptosis of the right eyelid of 6 years' duration (fig. 1).

The family history was noncontributory, and no relatives were known to have any similar difficulties. His past history was uneventful except for the usual childhood diseases and an occasional mild cold. The patient was unable to recall any details of his childhood ailments, other than they were the "usual childhood diseases."

The present illness began when the patient was 15 years old, at which time he sustained a minor laceration of the left eyebrow, with ptosis of the left eyelid apparently resulting therefrom. Two operations in that same year were unsuccessful in relieving the ptosis. In 1917, at 21 years of age, the patient enlisted in the U. S. Naval Reserve forces, at which time note was made of the ptosis of the left upper eyelid, and of a visual acuity of 18/20 in each eye.

In 1919, the patient was admitted to a hospital in New York, complaining of ptosis of the left eyelid, occasional blurring of vision, and diplopia on left lateral gaze. In addition to the ptosis of the left eyelid, he exhibited a bilateral restriction of the ocular movements in all directions, the left eyeball having an excursion of about 10 degrees from the central axis, and the right eyeball being restricted to about two thirds the normal range. Both pupils reacted slowly to light.

Refraction: O.D., +.50D. sph. = 20/20; O.S., +.50D. cyl. ax. 105° = 20/30. The physical examination was otherwise negative, as was the Wassermann test.

The course was uneventful until 1935, when, at the age of 40 years, he first noted ptosis of the right eyelid. From 1935 to the present, the

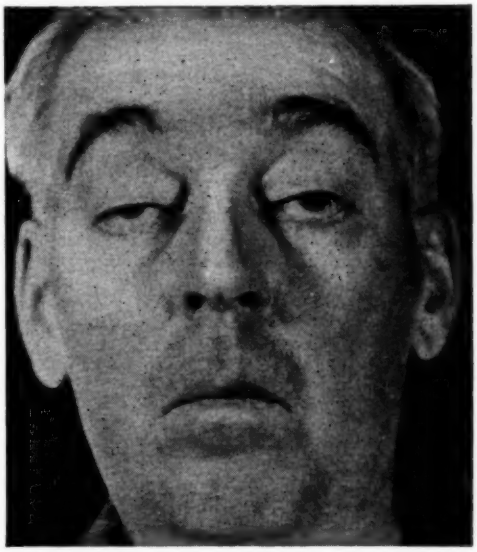

Fig. 1 (Fagin). A case of ophthalmoplegia externa.

restriction of ocular movements and the ptosis have slowly progressed.

At the time of admission to the Hospital, the patient was uncommunicative, lethargic, disoriented as to time, place, and person, and had fleeting delusions of being followed and watched on the ward by detectives. There was a small scar on the left eyebrow. The eyes exhibited bilateral ophthalmoplegia externa, including the levators, with ptosis and slight proptosis. The patient walked with his head bent far backwards in order to see through the narrowed apertures. The corneal reflexes were diminished. The pupils reacted normally to light and accommodation. The fundi exhibited accentuated cupping of the discs, with moderate pallor of the left disc. A small patch of retinal atrophy (one-half disc diameter in area) was present about one disc diameter below the left disc. The maculas, vessels, and disc margins were normal.

Visual acuity was O.D., 20/20—2; O.S., 20/100. The field of vision is shown on the chart (fig. 2).

The patient's speech was somewhat slurred and had a questionably bulbar paralytic quality that was difficult to evaluate because of the patient's lack of coöperation in the use of test phrases and even in ordinary conversation. How-

ever, his relatives stated that his speech had always been of that character. There was generalized weakness of slight degree, but no evidence of muscular atrophy. The weakness was not myasthenic in character and was not relieved by prostigmine. Sensation was intact in all modalities. Deep and superficial reflexes were normal, except for the diminished corneals.

tremor of the head and fingers diminished and finally disappeared. The patient became completely oriented and the delusions disappeared. The stolid facies, the slight weakness, and the ocular signs persisted unchanged, and the patient was discharged to his home on November 5, 1941.

At first, in view of the tremor, the stolid

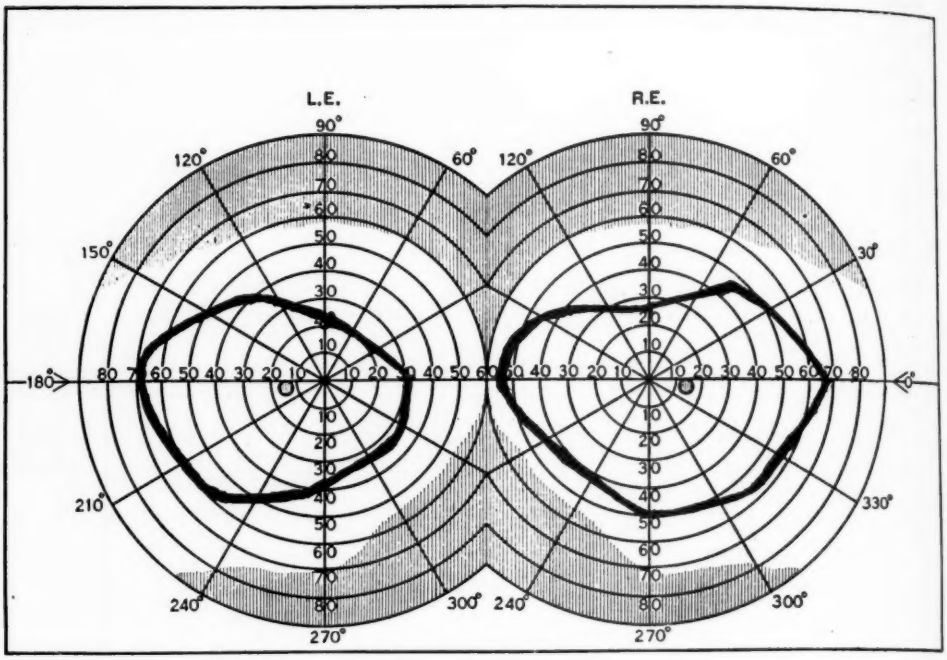

Fig. 2 (Fagin). Visual field.

Romberg's sign was questionably positive because of the generalized weakness. The patient's face was immobile and stolid, but there was no facial paralysis. The head and fingers exhibited a moderately coarse tremor, both at rest and on intention.

General physical examination was otherwise negative except for an apical systolic blowing murmur, not transmitted.

Laboratory findings were not very helpful. The urine, blood count, sedimentation rate, blood sugar, and N.P.N. were within normal limits. An X-ray examination of the chest was negative. X-ray-studies of the skull showed a dense cranial vault with a small amount of calcification about the sella turcica, probably due to atherosclerosis of the internal carotid artery. A spinal puncture revealed normal fluid as to cytologic, serologic, and pressure conditions. The blood Kahn test was negative.

The patient was uncoöperative in the extreme and refused practically all medication (strychnine, Rabellon, sedatives). With rest alone, the facies, and the general lethargy, it was believed that the patient's difficulties were probably due to a postencephalitic Parkinsonism, despite the absence of any history of encephalitis or influenza; and the temporary disorientation and delusions were attributed to a transient psychosis developing in the course of a somatic disease. However, the tremor was not characteristic of Parkinsonism, there was no loss of associated movements, no hypertonia, no sialorrhea. In addition, it would be most unusual for a postencephalitic state to manifest itself solely as an ophthalmoplegia for 30 years and then suddenly become reactivated. There is also the fact that the tremor and psychosis subsided spontaneously.

Reconsideration of the case, therefore, leads to the impression that it represents a chronic progressive ophthalmoplegia externa, due presumably to progressive nuclear degeneration. The transient psychosis probably represents a complication rather than a factor in the course of the disease. The case is reported because the

condition is sufficiently rare to warrant recording in the general literature.

Summary

1. A classification of the causes of chronic external ophthalmoplegias is presented.

2. A case of chronic progressive exophthalmoplegia externa is reported.

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EXPERIMENTAL TRANSPOSITION OF EXTRAOCULAR MUSCLES IN MONKEYS*

Role of the superior oblique

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The data presented in our previous paper[1] well illustrated the tolerance of the oculomotor apparatus to interference with the anatomic relationships between the extraocular-muscle tendons and the eyes. The experiments appeared to show, however, since incoördinated rotations did not result until four muscles and the superior oblique were manipulated, that the superior-oblique tendon and muscle had some special coördinating function that was destroyed when this muscle was detached from the globe. This impression could not be verified by the preliminary investigations, but the question arose, and further research was required in order to confirm or negate the possible assumption.

A series of experiments was outlined in which the physiologic effects of the superior oblique could be controlled and interpreted, yet effectual transposition of the muscles would be accomplished in

TABLE 1

Protocol of operations on the superior oblique in conjunction with transposition of other ocular-muscle tendons

Animal No.	Date	Eye	Muscles Operated on	Rotations
358	3/21/41	O.D.	Inf. and Med. Rec. Sup. and Lat. Rec.	Recovery
	9/23/41	O.U.	Tenotomy of Inf. Obl.	Recovery
376	1/10/41	O.D.	Sup. and Lat. Rec. Inf. and Med. Rec.	Limited but recovery of coördination
	6/20/41	O.D.	Tenotomy of Sup. Obl.	Same
	7/ 8/41	O.S.	Sup. and Lat. Rec.	Same
	9/23/41	O.S.	Med. and Inf. Rec.	Limitation O.D. makes judgment difficult
400	2/ 7/41	O.D.	Sup. and Lat. Rec. Inf. and Med. Rec.	Recovery
	5/ 2/41	O.S.	Sup. and Lat. Rec. Inf. and Med. Rec.	Recovery
	6/20/41	O.D.	Tenotomy of Sup. Obl.	Recovery
	7/10/41	O.S.	Tenotomy of Sup. Obl.	Recovery
390	7/11/41	O.D.	Tenotomy of Sup. Obl.	Recovery
	10/13/41	O.D.	Med. and Inf. Rec. Sup. and Lat. Rec.	Recovery
	11/ 7/41	O.S.	Tenotomy of Sup. Obl.	Recovery
	11/21/41	O.S.	Med. and Inf. Rec. Sup. and Lat. Rec.	No recovery
368	11/14/41	O.D.	Tenotomy of Sup. Obl.	Recovery
	11/28/41	O.D.	Sup. and Lat. Rec. Inf. and Med. Rec.	Recovery in darkness
367	4/25/41	O.D.	Med. and Inf. Rec.	Recovery
	5/21/41	O.D.	Tenotomy of Sup. Obl.	Recovery
	11/14/41	O.D.	Lat. and Sup. Rec.	Recovery
387	10/31/41	O.D.	Sup. and Lat. Rec. Tenotomy of Sup. Obl.	Recovery
	11/28/41	O.D.	Med. and Inf. Rec.	Recovery

A series of experiments was outlined in which the physiologic effects of the superior oblique could be controlled and interpreted, yet effectual transposition of the muscles would be accomplished in

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successive operations. These operations consisted of tenotomizing and resecting the tendon of the superior oblique either before or after other transposition operations had been made. In each case a recovery period of at least two weeks was allowed between successive operations. The operations performed and the results obtained are shown in table 1.

TABLE 2

RESULTS FOLLOWING TENOTOMY OF SUPERIOR OBLIQUE

Animal No.	Eye	Muscles Operated on	Rotations
390	O.D.	Sup. Obl.	No loss of coördination — impaired rotations down and in
	O.S.	Sup. Obl.	
368	O.D.	Sup. Obl.	Same
358	O.U.	Inf. Obl.	Same but impairment up and in

DISCUSSION

Maintenance and perhaps recovery of coördinated rotations, in so far as we know, depends upon vision and proprioception. That vision alone is not the sole mechanism in recovery is illustrated by the previously reported experiments in which the animals recovered from the transposition operations in total darkness. These experiments showed that interference with coördinated rotations did not occur so long as two muscles retained their normal anatomic and physiologic relationships. This is particularly illustrated by the recovery of coördination when only the four rectus muscles were transposed, whereas failure of recovery occurred when the superior oblique was also tenotomized. The two intact oblique muscles might be considered capable of giving the proprioceptive impulses necessary for recovery of associated movement, whereas one muscle only is inadequate. One can theorize further and consider that any two or more muscles would be sufficient to give the proper proprioceptive impressions to facilitate recovery from transposition or interference with the remaining muscles. Thus recovery occurred in animal 387 following simultaneous transposition of the superior and lateral recti and tenotomy of the superior oblique on the right eye. With coördination again established, operation of the medial and inferior recti was well tolerated. In a similar manner progressive transposition and tenotomy of the superior oblique was accomplished without loss of coördination in animal 367.

This analysis can be applied further to the animals in which four muscle transpositions had been accomplished with recovery of associated rotations, and in which at a later date the superior oblique was tenotomized (table 4). In this series it is shown that after recovery of rotations the superior oblique alone has no special function, although it with the inferior oblique may have been directly influential in initiating recovery from the

TABLE 3

RESULTS OF FOUR MUSCLE TRANSPOSITIONS FOLLOWING TENOTOMY OF SUPERIOR OBLIQUE

Animal No.	Eye	Muscles Operated on	Interval	Rotations
390	O.D.	Sup. and Lat. Rec. Inf. and Med. Rec.	3 mos.	Coördination
	O.S.	Sup. and Lat. Rec. Inf. and Med. Rec.	2 wks.	No recovery
368	O.D.	Sup. and Lat. Rec. Inf. and Med. Rec.	2 wks.	Recovery in darkness

first operation in which the four recti were transposed.

In agreement with expectations, tenotomy of the superior oblique (table 2) was tolerated without loss of coördination. We should now expect that transposition of the four rectus muscles should result in loss of coördination. The right eye of animal 390 was operated on and recovery resulted. The left eye was then operated on and recovery did not occur. The only difference in the operations was the interval between tenotomy and transposition, and the added disadvantage of having two eyes operated on instead of one. Vision was present during both recovery periods. Further to study the possible effects of vision, animal 368 was allowed to recover in total darkness. After two weeks the rotations were coordinated in this animal.

It is apparent that all the factors responsible for recovery of coördinated rotations are not yet known. The superior oblique alone does not appear to have any special coördinating function that cannot be ascribed to any other muscle. If proprioception plays a part in recovery, the impulses must originate in tissues other than the extraocular muscles and their tendons. One immediately thinks of the possibility of Tenon's capsule containing such endings, and it appears to the writers that there may be a relationship between the amount of surgical interference to Tenon's capsule and recovery of coördination. Further experiments will be made and reported concerning this phase of the work.

TABLE 4

RESULTS OF TENOTOMY OF OBLIQUE MUSCLES FOLLOWING FOUR MUSCLE TRANSPOSITIONS

Animal No.	Eye	Muscles Operated on	Interval	Rotations
358	O.D.	Inf. Obl.	6 mos.	Recovery
376	O.D.	Sup. Obl.	6 mos.	Recovery
400	O.D.	Sup. Obl.	4½ mos.	Recovery
	O.S.	Sup. Obl.	2 mos.	Recovery

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NOTES, CASES, INSTRUMENTS

FASCIAL SUSPENSION OF THE EYE

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One of the most serious complications following total excision of the upper jaw is the tendency of the eyeball to drop after it loses the support of the orbital plate of the maxilla. As observed by Fagge[1] this may be dislocated so that the affected eye looks down and not only becomes useless to the patient but causes much distress, owing to the resulting diplopia. The unfortunate individual ultimately becomes reconciled to enucleation. When removal of the orbital plate is essential, however, the tendency for the eyeball to drop may be greatly lessened provided the interal and external tarsal ligament, by which the suspensory ligament of Lockwood is supported, is not interfered with. My own observation has been that if the periosteal covering of the orbital floor can be preserved this greatly aids in the support of the eyeball and consequently lessens its tendency to drop.

Since the French surgeon Gensoul[2] first performed this operation more than a century ago, many prosthetic appliances have been made and plastic operations devised to overcome the downward dislocation, but with only partial success. Padgett,[3] in his recent book, declares: "Of the many attempts none have been completely successful." Up to the present time prosthetic appliances have ranged from cumbersome means of supporting the eye by inserting a prosthesis through an opening in the palate, to supports attached to spectacles molded to fit the concavity below the eye, producing sufficient compression to raise the eye to a normal plane. All of these are difficult to wear and in most cases too painful to be worn except for brief periods of time. Among the operations the most ingenious was that devised by König,[4] who attempted to prevent this complication by supporting the eyeball with a flap taken from the temporal muscle and suspended hammock fashion under the eye. A critical examination of König's technique reveals two faults: First, muscle tissue does not make a good supporting structure because the individual muscle fibers relax, allowing the hammock to sag with consequent dropping of the eye; second, only one end of the hammock is immobilized, the other moving with the mandible, thus affecting the stability of the support.

An attempt to overcome these faults prompted me to use fascia lata taken from high up on the thigh in order to secure the thinnest fascia. This was suspended hammock fashion and attached in the region of the outer and inner canthus by mattress sutures brought through and tied externally. The advantage of fascia over muscle is that as the healing process progresses and the hammock becomes attached to the tissues with which it is in contact the fascia contracts rather than relaxes, giving added rather than lessened support to the eyeball.

Two cases are presented:

Case 1. Osteoma of the Right Maxilla. The patient, aged 25 years, required total excision of the upper jaw. Figure 1 shows the position of the eye three years after

Fig. 1 (MacKenzie). Surgical results of fascial suspension in case 1, three years after the operation.

suspension. This procedure was supplemented with a vitallium restoration designed to replace the maxillary bone.

Case 2. Carcinoma of the Right Maxilla. The patient, aged 50 years, presented a large area of destruction involving the

Fig. 2 (MacKenzie). Result of fascial suspension in case 2, one year after the operation.

maxillary bone and half the cheek. The patient had previously received maximum doses of X-ray and radium therapy. Figure 2 shows the condition one year following suspension. The large opening in the cheek, which could not be closed by plastic means, was covered with a vitallium restoration attached to the spectacles.

Since operating on these patients a technique has been worked out whereby suspension by any means is no longer necessary in selected cases, for it is now possible to replace the orbital floor with a vitallium restoration with such anatomic precision that additional support is unnecessary. The eyeball is supported directly on the prosthesis. Hence the orbital compression remains unaltered, and there is no subsequent change in the dimension of the palpebral fissure, which widens or narrows as the compression increases or decreases, respectively. So far as I am aware, this has never been previously attained. In selected cases normal vision can be maintained with preservation of eyes that under similar circumstances have heretofore been sacrificed because of this distressing complication.

In a limited review of the literature I found no description of a fascial suspension of the eye.

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ACTINOMYCOSIS OF THE INFERIOR LACRIMAL CANALICULUS*

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Infection of the lacrimal canaliculi with organisms belonging to the higher bacteria has been known for a number of years. However, only a few reports have been made in the literature. The latest report was that of Elliot in 1941,[1] who added nine cases to those that have already been described. A total of about 20 cases has been reported in the United States.

The first clear description of a concretion in the lacrimal canaliculus was made by von Graefe in 1854.[2] He had seen some 10 cases. In 1869, Förster[3] described two more cases and made an interesting note on the possible route of the infection.

The disease is most commonly described as consisting of a firm, nontender lump in the lower lid, medial to the lacrimal punctum. The punctum itself is

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often reddened, elevated, and continually bathed in a small amount of mucopurulent discharge. Some epiphora may be present. The upper canaliculus may be involved but this is rare. The conjunctiva may also be infected by the actinomyces.

Report of case

D. K., a single, 19-year-old schoolboy, reported to the Stanford Eye Clinic on March 11, 1941, complaining that the right lower lid had been swelling off and on for the preceding year. He had been seen twice before in the Eye Clinic in 1935 and 1937 at which times the ocular examinations were negative except for a small degree of myopia.

The present examination of the right lower lid revealed a swelling of the tarsus just medial to the punctum. The punctal orifice was dilated, somewhat injected, and bathed in a thin, white discharge. The skin over the swelling was freely movable, while the conjunctival surface was mildly injected. The remainder of the palpebral and bulbar conjunctiva was normal. Pressure over the lump expressed a thin, white discharge from the punctum. Smears and culture of this discharge were taken and revealed gram-positive cocci, Staphylococcus albus (coagulase negative), and an anaerobic nonhemolytic streptococcus. No actinomycetes were found. Further examination of the eyes was negative.

The duct was patent to syringing, and X-ray examination, after injection with lipiodol, showed a normal unobstructed nasolacrimal duct without any outpocketing.

Examination by the Ear, Nose, and Throat Department revealed that the nasal septum was markedly deviated to the right and touched the middle and inferior turbinates. The tonsils were somewhat enlarged and injected. The general physical examination was negative, and a chest plate was essentially clear.

It was thought that the lesion was either a chalazion that had broken into the canaliculus or an actinomyces infection of the duct.

The lesion was treated with hot compresses and irrigated with 1:5,000 aqueous merthiolate. There was little change in the condition after two weeks of this treatment.

At this time the lump was incised through the tarsus, and a small cavity was revealed that contained a number of yellow green granules. The cavity walls were well curetted. The patient was given 0.5-percent sulfanilamide ointment to use three times a day at home. The incision healed quickly and all the symptoms were immediately relieved.

Six months later the lid showed only the scar of the incision and no recurrence of either tumor or discharge. The nasolacrimal duct was patent to syringing.

The granules removed from the lacrimal-duct cavity revealed in wet specimen an organism of the actinomyces group. After culture in semisolid agar media for five days, cover-slip preparations showed rosettes with clubbing, typical of actinomyces, and stains showed the typical bacillary and threadlike forms.

Although all reported cases have cleared up following evacuation of the concretions or granules from the lacrimal-duct tumors, we believed that since Dobson, Holman, and Cutting[4] reported good results from the use of sulfanilamide in the therapy of actinomycosis in other body regions, we might speed the recovery by use of sulfanilamide directly to the lesion.

In both the ophthalmologic and bacteriologic literature there has been a great deal of confusion concerning the name and classification of the organism causing this condition. Many authors have mentioned it and the number of titles un-

der which these case reports have been written will testify to it. Streptothrix, leptothrix, and actinomyces have been the most commonly confused terms. Leptothrix can be easily identified from the others, and therefore should cause no confusion. There are sufficient grounds for confusion between streptothrix and actinomyces.

Emmons[5] has made an excellent summary of the history of actinomyces. The first description of an organism of this group was given by Cohn in 1875 from organisms obtained from the tear duct of a human eye. Cohn named his organisms Streptothrix försteri, after the eminent ophthalmologist. Then, in 1877, Harz described an organism obtained from a case of "lumpy jaw" in cattle. An aerobic form of the Harz organism was first cultured by Bostroem in 1891, but most American investigators now consider this to be a saprophytic contaminant. However, in the same year Wolf and Israel obtained a microaerophilic strain that is always found in the actinomycotic diseases in animals and man. This form almost always is found as a club-bearing granule. Clubs are not diagnostic of actinomyces, for other organisms have them and some species of actinomyces do not have them. They are considered to be formed by the interaction of the host and the parasite.

The term streptothrix, having once been introduced for this higher bacterium and particularly in as much as it was originally associated with a lacrimal-duct lesion, has remained firmly placed in the literature. However, it is entirely invalid since it was previously used by Corda in 1839 for an entirely different group of fungi. This group still is recognized by this name. The Committee of the Society of American Bacteriologists on Characterization and Classification of Bacterial Types and Bergy's "Manual of bacteriology" recognize actinomyces as the correct name. Kipp[6] in 1902 states that Schroeder in 1894 expressed the opinion that Streptothrix försteri was identical with the actinomyces. Ruys[7] in 1935 stated: . . . "following the opinion of Lieske, Topley and Wilson, and other authors, streptothrix and actinomyces are now considered as all belonging to the same genus actinomyces. Formerly the presence or absence of drusen was considered satisfactory for ranging a strain to the genus actinomyces or streptothrix. As, however, this phenomenon depends on the reactions of the host and not on the peculiarities of the parasite, this is not sufficient evidence for making two genera. Then in most instances the concretions in the lacrimal ducts described in the literature are caused by actinomyces. It is not certain whether leptothrix also plays a role."

The exact mode of infection has never been proved. The actinomyces organism is microaerophilic. Some claim that it comes from air-borne spores that develop into the anaerobic form on becoming located in the canaliculus. Emmons[8] claims that this is a result of the misconception introduced by Bostroem through his aerobic saprophytic form. Förster[2] in his 1869 report mentioned the possibility of transferring the organism from the mouth to the eyes through the medium of the fingers, as it seemed rather common in his time to wipe the eyes with saliva. The Actinomyces seems to be a fairly common inhabitant of the mouth, and Emmons[8] found that in 100 consecutive tonsillectomies actinomyces was present in 47 percent of the tonsils. Since practically all of these must have been of very low virulence and might be the source of the infection of the eye, the extremely low virulence of the canaliculus infections can be understood.

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PLASTIC SUTURE PLATES FOR THE CLOSURE OF WOUNDS IN CERTAIN OPHTHALMIC OPERATIONS*†

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New York

Plastic plates have been found useful in closing small wounds of the cornea and conjunctiva, in operations on the ocular muscles and in plastic operations, to lessen scarring and loss of tension from sutures.

The quadrangular plates are made of lucite‡ or plexiglass,§ 5 mm. by 2 mm. by 3 mm. in size; two holes are drilled for the passage of atraumatic needles. The edges are slightly rounded (fig. 1A). For larger surfaces, where traction can be made at a distance from the wound, plastic discs (fig. 1B) are recommended. Various sizes may be obtained.

The advantages are that plastic plates

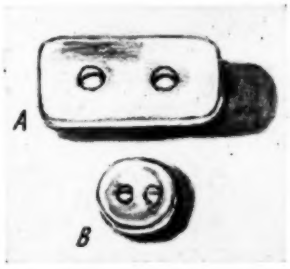

Fig. 1 (Berens). Plastic suture plates for the closure of wounds in certain ophthalmic operations: A, quadrangular plates; B, circular plates.

(a) will not irritate the tissue,[1] (b) conform slightly to the tissues, (c) do not absorb serum or pus, and (d) are less expensive than gold plates.[2]

35 East Seventieth Street.

* Made by V. Mueller and Company, Chicago, Illinois.

† Aided by a grant from The Ophthalmological Foundation, Inc.

‡ Lucite is manufactured by E. I. du Pont de Nemours Company.

§ Plexiglass by Rohm and Haas Company, Philadelphia, Pennsylvania.

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INTRANASAL DRAINAGE FOR CURE OF CHRONIC TEAR-SAC INFECTION*

NEW TECHNIQUE AIDED BY ELECTROCOAGULATION SO SIMPLIFIED AS TO BE AN OFFICE PROCEDURE

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Brooklyn, New York

The patency of intranasal bony openings is best maintained by the use of electrocoagulation.[1] Openings made by surgery alone often close, resulting in failure. For this reason, I have employed electrocoagulation in a new technique for the cure of chronic tear-sac infection. This technique is accurate and direct, with tissue destruction so minimized that the whole procedure can be performed in the office, thus avoiding the cost and inconvenience of hospitalization.

Local anesthesia is used. A dental root-canal probe, via the canaliculus, is pushed through the bottom of the tear sac into the nasal cavity as a guide. Four or five instruments are fashioned from various sizes of stiff steel wire, the ends bent at 90 degrees and sharpened bluntly. The smallest hook is slid along the back of the guiding probe, penetrating the lateral nasal wall. Palpating with the forefinger of the free hand at the inner canthus, the operator feels for movement of the dull point of the largest hook used last. Under guidance of touch, the infected tear sac is opened to its full extent. Similarly, after insulating all but the bent end of this hook, fragments of the torn sac, its contents, and the bony opening to it are electrocoagulated. The tear sac is copiously irrigated through the newly formed opening into the nose. There is scarcely any bleeding.

This technique is best adapted for narrow noses; fracture, tuberculous, and congenital syphilitic cases where there is marked new bone formation. Among its other advantages are: (a) Removal of enlarged tip of middle turbinate unnecessary; (b) preliminary resection of nasal septum reduced to lowest number; (c) smallness of final opening prevents blowing air or infected matter from nose to eye. In acute cases, when serious complications threaten, this technique requires the least surgical interference to start drainage and seals vascular channels almost simultaneously.

The technique here briefly outlined is the first that employs hooks of simple construction, aided by electrocoagulation of the tear sac and the bony opening to it, to achieve intranasal drainage and permanent cure of chronic tear-sac infection.

433 Eastern Parkway.

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* Abstract of paper read by invitation before the New York Society for Clinical Ophthalmology on March 3, 1941. Complete article in Archives of Ophthalmology, April, 1942.

† Assistant attending at the New York Post-Graduate Medical School and Hospital, Columbia University.

‡ Following excision of the sac, when tearing persists, this technique can be adapted to form a new pathway into the nose for tear conduction.

§ Retrograde diathermic catheterization may be all that is required to cure chronic catarrhal cases of tear-sac infection.

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CASE OF CONJUNCTIVITIS CAUSED BY STAPHYLOCOCCUS AUREUS GIVING A STRONGLY POSITIVE COAGULASE REACTION

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Mrs. M. L., aged 30 years, a housewife, presented herself on November 22, 1941. She had previously been treated by her family physician for one week for an inflamed conjunctiva and phlyctenules around the corneal margin, which still persisted on presentation. The lids were dry. The patient was given a sodium biborate wash and holocaine and epinephrine ointment. The phlyctenules were touched up with mercurochrome. Two days later there was much improvement.

On November 27th, the right eye showed marked exacerbation. The lids were very red and swollen, the conjunctiva much inflamed, and the patient was in great pain. One-fourth percent atropine was instilled and merthiolate ointment placed in the eye.

On November 29th, the left eye was much inflamed, resembling inclusion-body blennorrhea. Scrapings were made from the upper lid of this eye and smeared on a slide. They were stained with iodine and also with Giemsa. Search failed to reveal any inclusion bodies. Numerous pus cells were seen everywhere and only a few epithelial cells. Everywhere numerous, scattered, gram-positive cocci were present. Culture was made on a blood-agar plate.

Five-percent sulfathiazole ointment was ordered for application night and morning, and 5-percent sulfathiazole solution to be instilled every two hours. The patient was not seen again until December 6th, when both eyes were entirely normal, and, according to patient, had been so for several days.

The culture on blood agar yielded a pure growth of Staphylococcus aureus, colonies about two to three millimeters in diameter, with a zone of hemolysis about a millimeter wide around each colony. Staphylococcus being such a ubiquitous organism and being so frequently found in so many conditions, ocular and otherwise, the coagulase test of Chapman, Berens, Peters, and Curcio was made (Jour. Bact., 1934, v. 28, p. 343). One-half cubic centimeter of citrated normal human plasma was put in a test tube, then with a loop a quantity of the growth on the blood-agar plate was emulsified in it. The test tube with the plasma and Staphylococcus was placed in an incubator. During the first hour the plasma formed a firm clot. This test is considered rather specific for pathogenic staphylococci, and the conclusion drawn is that the entire process was caused by a virulent hemolytic Staphylococcus aureus.

Four adults and one child were living in the same household, and none of them contracted the same disease, although when a possible blennorrhea was suspected, the child was removed from the home.

Remarks. Virulent staphylococci can cause inflammation resembling that of inclusion-body blennorrhea. In this case sulfathiazole used as solution and in ointment form acted as a specific.

* Died on January 18, 1942.

BILATERAL EXUDATIVE RETINITIS IN A CASE OF CEREBRAL HEMORRHAGE; CHRYSOTHERAPY WITH RECOVERY*

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AND
MAXWELL L. GELFAND, M.D.
New York

The use of gold compounds in ocular therapeutics is recent and uncommon. It seems to have been initiated with the ionization experiments of Abramowicz[1] in 1927. Since then several reports of good results obtained with chrysotherapy have appeared, including two in the American literature (Benedict and Goeckerman[2] and Koenig[3]). The following case is reported for two reasons: first, because of the unusual type of bilateral ocular lesion accompanying an intracranial hemorrhage and, second, because of the unexpected therapeutic result apparently obtained with gold sodium thiosulphate.

C. B., a white male aged 40 years, was first seen by one of us (M. L. G.) on April 20, 1938. His chief complaint was dyspnea and swelling of the ankles. Examination disclosed that the patient was a hypertensive and that he had left cardiac enlargement. Laboratory studies of the blood and urine were negative. The proper regimen was instituted and the patient got along comfortably for some time. On November 8, 1938, while hurrying home because of a severe headache he collapsed in the street and was taken to a nearby hospital. Here it was found that he had an intracranial hemorrhage with right-sided hemiplegia, bloody spinal fluid, and systolic blood pressure of 240. He recovered consciousness on the fifth day and immediately noted that his vision was limited to bare light perception in both eyes.

He was seen in ophthalmologic consultation (by S. A. F.) on January 11, 1939. External examination of the eyes showed normal lids, conjunctivas, and corneas. Both eyes were turned slightly to the right. The tension (Schiötz) was 19 mm. Hg in the right eye and 17 mm. in the left. There was no pain and not the slightest evidence of uveal involvement. The pupils were slightly though evenly dilated and reacted somewhat sluggishly to light. There were no apparent muscular anomalies. Vision was limited to light perception with poor projection in both eyes. With the right eye no light was seen above and with the left eye no light was seen above nor to the right. On dilatation and ophthalmoscopic examination both lenses were found to be clear. Both vitreous chambers were so packed with huge opacities, apparently the result of exudative and hemorrhagic processes, that only the lower fundus reflex was visible in the right eye. In the left eye there was a better fundus reflex, but no details were visible in either eye. What could be seen appeared to be a tremendous proliferating retinitis in both eyes, with occasional hemorrhagic areas.

Dionin and hot compresses were prescribed and also daily homatropine, as the patient preferred the increased light which this permitted. Potassium iodide was given internally. He was under observation at home for about six weeks, then, on February 21, 1939, was well enough to come to the office. The ocular status at this time was found to be essentially unchanged. Slitlamp examination was done for the first time and disclosed an absolutely clear cornea, aqueous,

* Read before the New York Society for Clinical Ophthalmology, January 5, 1942.

and lens. The vitreous and fundus pictures were unchanged. At the end of May the general condition, including vision, was unchanged. The vitreous was full of opacities and the fundus, as much of it as could be seen, was almost completely covered with huge consolidating retinal exudates. It seemed obvious that, visually, the patient was getting nowhere and that barring the unusual he would apparently remain permanently blind.

On June 6, 1939, chemotherapy in the form of gold sodium thiosulphate injections was instituted. The patient received a series of 23 weekly injections intravenously (he would have received 24 but one of the ampoules was broken). Ten mg. were given the first week by way of exploratory procedure in order to see how the patient would react. The next six weekly doses consisted of 25 mg. each, and the remainder were of 50 mg. each. At no time during the course of treatment was there any untoward reaction.

After the third or fourth injection the patient stated that he could see better, although visual tests did not confirm this. After the sixth injection, however, we were surprised and gratified to find that vision in the right eye had improved so that he could count fingers at 3 feet and in the left eye to 20/400. After the ninth injection the vision was 20/40 in the right eye and 20/40 in the left eye improvable to 20/30+2 with a +0.50 D. cyl. ax. 120°. A few fine tortuous vessels could now be made out in the fundus among the exudative patches. From then on there was slow but constant improvement so that about two weeks after the final injection on November 23, 1939, the vision was 20/20 in the right eye and 20/25 in the left improvable to 20/20 with a +0.50 D. cyl. ax. 120°.

Fields done on the same day with a 0.7-degree white object showed the form field in the right eye to be normal above but contracted to the 40-degree isopter elsewhere. The left eye was practically normal except for a cut in the lower field to the 25-degree isopter. The patient was able to read Jaeger 2. On February 5, 1940, the condition was approximately the same. The vitreous still contained many exudates in both eyes and the retinas were still covered with many proliferative patches. But the patient was quite happy as he had normal vision and could read without difficulty.

In November, 1940, the patient died of coronary occlusion.

The cause of the marked exudative and hemorrhagic phenomena noted in this case is speculative. One possible explanation is a disordered capillary permeability caused by obstruction to venous outflow as a result of cerebral edema and increased intraocular pressure. This may have resulted in a massive outpouring of exudativa vascular products into the retina with rupture through the limiting membrane into the vitreous. We are at a loss to explain the excellent and totally unexpected result obtained with the gold sodium thiosulphate. Nor are we overlooking the possibility that the visual improvement may have been purely coincidental and that it might have occurred anyhow. However, this is a case in which there seemed to be not the slightest improvement for seven months and in which marked improvement did appear after the administration of six intravenous injections of a drug. It would seem at least as justifiable to suppose that the improvement was due as much to the treatment as in spite of it.

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A FLEXIBLE TIP FOR THE GIANT MAGNET*

LELAND F. CARTER, M.D.
Detroit

The use of the giant magnet for removal of foreign bodies from the anterior chamber often presents difficulties, some of which may be listed as follows: (1) Poor visibility; (2) difficulty in maneuvering the magnet tip to the proper angle; (3) magnetic pull on a strabismus hook or other instrument, used as an extension of the tip, hinders freedom of movement; (4) danger of traumatizing the globe as a result of any of the above-mentioned encumbrances.

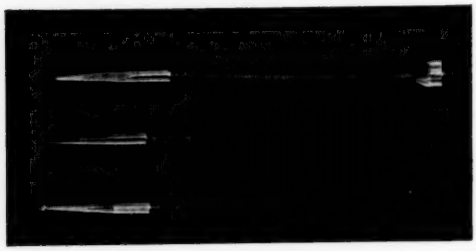

Fig. 1 (Carter). A flexible tip for the giant magnet.

The flexible magnet tip was found to have none of these disadvantages when used in operating upon a fairly large number of consecutive patients presenting cases of intraocular foreign body, one of which was removed by the transcleral route.

The instrument is fitted with either a screw-in or a plug-in attachment to replace the regulation tip in the core of the magnet. Acting as an extension from the magnet is a five-inch flexible steel-coil spring, to the free end of which is attached the tip. Three screw-on, interchangeable tips of varying degrees of magnetic pull are available.

613 David Whitney Building.

* Presented at the meeting of the American Academy of Ophthalmology and Otolaryngology at Chicago, 1941.

SOCIETY PROCEEDINGS

EDITED BY DR. RALPH H. MILLER

CHICAGO OPHTHALMOLOGICAL SOCIETY

February 17, 1941

DR. RICHARD C. GAMBLE, *president*

CLINICAL MEETING

(Presented by the Department of Ophthalmology, Northwestern University.)

GLAUCOMA SECONDARY TO IRIS ATROPHY

DR. HELEN HOLT said that the patient, a woman aged 65 years, had had three attacks of severe orbital pain radiating over the right side of the head, associated with nausea and vomiting, during the year preceding her admission to the clinic. The eye would become very red, the vision blurred, and the pupil enlarged. When first seen in January, 1941, the left eye was normal in all respects. Vision of the right eye was 20/200, very blurred, insufficient for any Jaeger reading. The pupil was irregular, more widely dilated at the 12-o'clock position, and about 6 mm. in diameter. The cornea was hazy; the slitlamp showed bedewing and fine pigment precipitates on the posterior cornea. The pigment on the posterior surface of the iris was absent in irregular patches in the superior and upper nasal portions. The pupil failed to constrict in the upper portion. The disc was normal; no cupping was evident. There were arteriosclerotic changes in the vessels. Gonioscopic examination of the right eye showed a chamber of normal depth with pigment in the angle. A nasal step was demonstrable in the first field taken. The tension (Schiötz) was R.E. 23.5 mm. Hg, L.E. 20.5 mm. Eserine 0.2 percent was prescribed. The pupil constricted only partly. Vision improved to 20/30 within one month and the fields became full. The patient felt more comfortable than she had for a year. It was concluded that the glaucoma was secondary to the iris atrophy.

RETINITIS PIGMENTOSA

DR. HELEN HOLT said that F. H., a woman aged 35 years, had had poor vision since childhood, associated with difficulty in getting about in the dark. External examination was negative, but the fundus showed typical changes of retinitis pigmentosa. The peripheral fields ranged from 10 to 15 degrees in each eye. Photographs of the retinal vessels were made under normal conditions and following prostigmine, which induced no changes. Using the biophotometer to test dark adaptation the patient took 35 minutes to perceive the strongest illumination and never did see the more diminished light.

Bilateral cervical sympathectomy was performed, following which the patient was certain that her vision was improved and that the twilight periods were not so long. Objectively the condition remained the same; vision 20/200, J. 2, each eye; the caliber of the retinal vessels remained unchanged. The patient perceived the first light on the biophotometer in seven minutes, a marked improvement. Whether this is a temporary change or not will be determined by future tests. Perhaps greater improvement could be hoped for in less advanced cases.

MALIGNANT ULCERATION OF THE LOWER LID

DR. SPEAR said that 11 years ago this patient, a woman aged 68 years, had an ulcerating lesion at the outer angle of the left eye which was treated with radium. Complete healing took place, with scar-

ring and distortion of the tissues. Four years ago there was recurrence of the lesion, which has gradually increased in size and has received no treatment. An ulcerated area 3 cm. by 2 cm., with hard nodular irregular margins, extended from the center of the left lower lid to the outer canthus and around to the brow, which was drawn down. The lid tissues were entirely destroyed in this area and the tissues fixed to the bone. The lower fornix was invaded by the growth, but the eyeball was not involved and was freely movable. The lens was normal. Intense radium and X-ray treatment has been initiated and will most certainly damage the eye. Comparison will be made in the future when the lesion is healed so that the possible effect of the treatment on the eye can be demonstrated.

Retroretinal tissue from the choroid in Kuhnt-Junius disciform degeneration of the macula—anatomical study*

Dr. E. V. L. Brown said that the Kuhnt and Junius monograph focused world attention upon this interesting condition in 1926. Their studies led them to believe that the disease is primarily one of the retina. But none of their eight cases came to anatomic study and recent studies indicate that the choroid is the primary seat of the disease. Some 18 such reports are now at hand, 3 before and 13 since 1926, including the one by our own members, Drs. Gifford and Cushman in 1939. The connective tissue and hemorrhage between retina and choroid has been shown to come from the choroid, through breaks in Bruch's membrane. Such breaks have been found in 13 of the eyes studied (Axenfeld, Woelfflein, Paul, Behr, Hanssen, Vogt, Braun, Rintelen, Verhoeff and Grossman, Gifford and Cushman, and Sandoz). They have not been found in five other eyes (Hegner, Elschnig, Seefelder, Hanssen, and Verhoeff and Grossman). Hegner, Seefelder, and Hanssen, however, do not specifically state that they made serial sections. Furthermore, long stretches of vascularized tissue between choroid and retina have been reported anterior to the equator in otherwise normal aged eyes (Reichling and Klemens). Wolfrum found tissue between the lamina elastica and the basal membrane in older people, 30 years ago, but his observations have received scant attention. The present case is presented merely because serial sections, lacking to date in a good many cases, fill out the anatomic picture to a certain extent.

The patient, a woman aged 74 years, presented considerable opacity of the lens and a large central chorioretinitic lesion with glistening dots in its lower portion, reduction of vision to counting fingers at two feet, recognition of large objects in a small part of the central field, and a loss of the upper nasal field.

An 8- by 9-mm. mass of new tissue about 0.1 mm. thick lay between choroid and retina. Anterior to and continuous with this "basal tissue" was a "mound" of tissue, temporal to the fovea, or rather several mounds merged into one. The mound covered nearly half the expanse of the basal tissue from side to side and over a quarter of it from above down, and the mound was five times as thick as the basal tissue. The outer layers of the retina were destroyed in front of the mound and fused to it by glia. The forepart of the basal lesion consisted of pigment epithelium proliferated into a "connective" tissue. The back portion (next to the choroid) was genuine richly vascularized connective tissue, and it continued directly into the tissue of the mound; the latter was full of collapsed obliterated new-

* Résumé of an article published in the Arch. of Ophth., 1940, v. 23, June, pp. 1157-1168.

formed vessels, but was notably free from pigment. A tiny vesicle lay beneath the raised pigment epithelial layer just temporal to the mound. It was full of proliferating unpigmented pigment epithelium and had a connection with the choroid through a small break in the lamina vitrea near the mound. Other breaks were numerous (54), and through them blood, fibroblasts, capillaries, and good-sized vessels made their way from the choroid into the basal tissue and mound. Hemorrhage was abundant on all sides of the mound.

As to pathogenesis, the essayist held that definite breaks in the lamina basalis were not really necessary, theoretically, to explain such retroretinal tissue. Changed secretion or marked increase in normal secretion from the choroid and its accumulation into a bleb or vesicle of stagnant albuminous fluid beneath the pigment epithelium would be sufficient to excite proliferation of the pigment epithelium. Primary disease of the lamina vitrea could of course do this by itself, as held by Behr. The large number of breaks found in the lamina vitrea in the case presented would obviously facilitate both irritation and proliferation of pigment epithelium and the development of large amounts of collagenous tissue behind the epithelial tissue, from hemorrhage, new-vessel walls, and so forth. Finally, the process could well have periods of exacerbation and recurrence, as noted clinically.

Discussion. Dr. Sanford Gifford said that in the literature, as he and Dr. Cushman had gone over it, there were about nine cases that came to section in which defects were found in the lamina vitrea. Their case and that of Dr. Brown, would make a total of 11. There were three in which no breaks were found, but Hanssen's case showed a change in the lamina vitrea which would bear out Dr. Brown's idea that it was not necessary to have a complete break.

It was interesting to note that in nine cases of Kuhnt-Junius disease there were associated drusen in unusual number and size. It was not thought that the drusen were the cause of the disease, but that they indicated a disease of the lamina vitrea and that the changes were more likely to occur with the occurrence of drusen. In only one case in the literature had any connection been traced between the hemorrhages and the retinal vessels. The hemorrhage usually comes from new-formed vessels with very delicate walls, deriving from the choroid. A few eyes may show a flatter lesion with no hemorrhage, and it may be that the new vessels have not yet broken through. Later on with growth of the new vessels, hemorrhages are apt to occur.

It was also noted that these patients do not have any more arteriosclerotic changes than are observed in other people of the same age. The original theory was that the condition was due to arteriosclerosis, but it is now felt that the disease is primarily in the lamina vitrea and the choroid. There is no treatment. All that can be said is that the patients will not become blind.

Dr. Louis Bothman remarked that one of Dr. Brown's slides showed a vessel herniating through the lamina vitrea of the choroid. This explains something often observed clinically. Frequently, after it has been present for a long time, one wonders why an area appearing like a hemorrhage does not become absorbed, inasmuch as other hemorrhages disappear. This vessel hernia or small angioma is probably the explanation. The ones that do not disappear are either hernias of choroidal vessels or angiomata. In the fundi of diabetic eyes, Friedenwald showed similar changes that looked like tiny hemorrhages. The punctate mul-

berry-colored lesions, which persist in Kuhnt-Junius disease, are also small hernias or angiomata.

Dr. Robert Von der Heydt presented kodachrome slides that showed a typical though different picture from those shown by Dr. Brown. They present large circinate lesions possibly due to hemorrhage; the hemorrhages are retroretinal. One picture showed a far-advanced Kuhnt-Junius lesion in the patient's one eye and the very incipience of the process in the other.

Dr. Bertha Klien said that the large number of breaks in the lamina basalis in Dr. Brown's sections was most remarkable, and raised again the question of the relationship between the Kuhnt-Junius degeneration and angioid streaks. Clinically, there were no angioid streaks in Dr. Brown's case. This supports the view that something more than a crack in this membrane is necessary to produce the clinical picture of angioid streaks; that is, a degeneration which renders the membrane opaque, so that the breaks become visible by contrast.

In angioid streaks there is a diffuse degeneration of the elastic portion of this membrane as part of a systemic disease that has its first manifestations in eye and skin. In the Kuhnt-Junius degeneration it would seem to be a more circumscribed process due to local causes in the eye, resulting perhaps in only an altered permeability of the membrane. That this is, however, not always limited to the posterior polar region, as one might expect from the clinical picture, is beautifully demonstrated in Dr. Brown's sections.

The comparison of these two diseases leads to speculation about the possible varieties and types of degenerative processes affecting the lamina basalis, those that render the membrane opaque and thus permit clinical diagnosis of a simple defect, and those that eventually cause dehiscences in the membrane, which are not visible clinically until a reactive process from the surrounding tissues sets in, as in the disciform degeneration.

Dr. E. V. L. Brown, in closing, said that some sclerosis of the choroidal vessels was found. It is hard to get a good index of what constitutes a sclerosed vessel. If the wall of the vessel was two times as wide as the lumen, it was considered sclerosed. Using this yardstick, twice as many sclerosed vessels were found in the choroid behind this tissue as elsewhere. There were no sclerosed vessels in the retina, in the optic nerve, or in the uvea. This local process could account for changes in secretion and perhaps for changes in the lamina vitrea.

Some earlier reports of Coats's disease simulated the mound formation very closely. But Coats's disease is an external retinitis and not a choroiditis, and is often located far from the macula.

Detachment of the Pars Ciliaris Retinae. A Clinical Finding of Differential Diagnostic Value in Retinal Detachment

Dr. Bertha A. Klien presented a paper on this subject.

Discussion. Dr. Beulah Cushman said that after reading Dr. Klien's paper she went over the pathologic material at Northwestern and found seven eyes in which there was separation of the ciliary unpigmented epithelium, none of which contained any clinical changes that would suggest such a lesion on examination, but they were all cases of late tumor or severe injuries.

Four were malignant melanomas of the uveal tract, two of the ciliary body and two of the posterior segment. Those of the ciliary body show the separation of the unpigmented epithelium in the area opposite the tumor. In those of the posterior segment, the separation of the

retina was continued forward with separation of the unpigmented ciliary epithelium over the pars plana to corona ciliaris.

Three were cases of perforating injuries: One followed preliminary iridectomy in which the lens capsule was ruptured and an endophthalmitis phacogenetica developed. In one an anterior staphyloma had developed in the region of the perforating injury, and on the side opposite, the unpigmented ciliary epithelium was separated. The third was in an eye which was removed immediately following an extensive perforating injury. On the side exhibiting the separation of the retina the unpigmented ciliary epithelium was separated by a diffuse hemorrhage.

Salzmann states that the union of the unpigmented ciliary epithelium with the pigmented epithelium is a great deal more firm than is that of the retina with the pigment epithelium of the choroidea, and that this union is affected by a cement substance like that uniting the individual ciliary epithelial cells. It would seem, therefore, that the separation of the unpigmented ciliary epithelium would take place with a slow continuous pressure, as from hemorrhage or subretinal fluid seeping forward from a tumor, since artificial and postmortem separations usually occur at the border of the retina.

Morphology and Function of the Scleral Vessels

Dr. P. F. Swindle presented a paper on this subject.

Discussion. Dr. Michael Goldenburg was of the opinion that Dr. Swindle had made a real contribution to the anatomy and physiology of the ocular circulation. His contributions to ophthalmology are only a small part of his work as a physiologist. The slides shown cannot possibly do justice to his specimens; they should be examined with a low-power binocular microscope to better trace the many anastomoses that take place throughout at various levels and between the different levels. One can then appreciate the circulation as a unit and can better understand that when pathologic changes occur in a sector of the unit, they must influence the entire unit in proportion to the changes.

Dr. Swindle's observations would seem to be of value in the glaucoma problem. The petechial infarcts taking place in the uveal circulation may be few or many, and may coalesce and form relatively large infarcts and create diminished fluid escape and, in time, an elevation of intraocular pressure. Birnbacher and Czermak called attention years ago to the pathologic processes in and around the vortex veins in glaucoma. Koster ligated all four vortex veins, and a very high pressure soon followed, the pupil dilated, and the anterior chamber became shallow. After about three weeks, however, the eye returned to normal except for the anterior peripheral iris synechia. When two or three vortex veins were ligated, the area drained by these vessels alone was involved, thus demonstrating the collateral-circulation possibilities. The venae vestigia oculi with their open mouths in the drainage angle, the vestigial remains of the embryonic circulation before the anterior chamber is formed, must recall to us the microscopic findings in buphthalmos and should cast some light on that type and the juvenile type of glaucoma. In these types may there not be a lesser or total absence of these openings in the drainage angle, the result of a disturbed evolution of the embryonal tissues located here? He also called attention to the long, thin, scleral arteries having the property of extreme constriction, which should make one think of the vasospasm described in essential hypertension with

damage to the endothelium, hyperplastic thickening of the media, and diminution of the lumens of these vessels thus disturbing the circulation. This which may be a factor in the glaucoma cycle, especially in the noncongestive type.

Whether one prefers Dr. Swindle's classification of the scleral blood vessels, which he refers to as the circle of Hovius, or the more popular classification, is not really important. It would seem that these classifications are only the application of different terms to the same thing, probably the result of different interpretations for the same structures. The venae vestigia oculi of Swindle, the inner canals of Sondermann, the collector veins of Maggiore, the collector channels of Theobald are terms applied to different parts of the vessels present in this region. It is very evident that a rich blood supply does exist, and that a free anastomosis between these vessels is present not only at the same level but between the different levels. The ligation of the vortex-veins experiment now takes on a different meaning, the possibility of the reëstablishment of the circulation by a collateral circulation and decrease of pressure in a normal eye, whereas the petchial infarcts referred to would mean the gradual pathologic groundwork for an eventual circulatory episode that might not be overcome and could result in stasis and pressure.

Dr. Georgiana Theobald said that it is of great interest to study and compare the eyes of the animal kingdom—to note the stages of development from the simplest organ of vision to the perfected eye of man. Even though he states that he cannot appreciate any fundamental difference between the human and the dog's eye, certainly the essayist must see some difference in the eyes of various animal groups. Comparative anatomy shows that the development of organs depends on functional use; certainly this is true of the eye.

The early anatomists attributed to the human eye what they discovered in the animal eye. In 1716 Hovius described a large venous circle in the scleras of certain animals parallel to the iris insertion. In 1781 Fontana described spaces in the anterior-chamber angle of cow eyes. Probably due to some error of manipulation he described the development of this tissue as a canal. In 1830 Schlemm discovered a circular vessel in a human eye, which now bears his name. He cautioned against confounding it with Fontana's canal. According to Troncoso, Schlemm's canal first appears in primates and is not found in carnivores nor ungulates. Because of these essential differences between the eyes of primates and the lower animals, the circulation of the anterior portion is also different.

For years the access of aqueous from the anterior chamber to the canal of Schlemm has been the subject of great discussion. Physiologic experiments prove and disprove open connections, depending on the experimenter. In 1933 Sondermann, in cutting serial sections, described openings from the intratrabecular spaces into the canal of Schlemm itself.

Dr. Swindle says: "The arteries of the circle are capable of constricting so strongly that their lumina become too small to conduct the particle of injected India ink. This is true shortly after eserine has been dropped into the conjunctiva." From all data found in the literature, however, eserine causes a vasodilation. The vessel could constrict because of an irritation by the India ink. Also, constriction of the pupil is due to the action of eserine on the neuromuscular junction between the third nerve and the sphincter pupillae, and not because the tortuous radial arteries of the iris become relatively nontortuous. The iris has never

been regarded as erectile tissue. It has been shown that when the third nerve is paralyzed the pupil does not constrict when eserine is instilled into the conjunctival sac. This shows that the constriction is on a nerve-muscle relationship, and not a vascular one.

Atropine has a threefold action on the eye. By paralyzing the intraocular muscles it puts the tissues of the eye to rest. It causes a vasodilation, and it dilates the pupil by paralyzing the junction between the third nerve and the sphincter.

According to Duke-Elder, (volume 1, page 139) the muscular coat of the arteries almost ceases after the arteries pierce the sclera (Bartels, 1905; Kümmel, 1908; Fuchs, 1929). In the choroid and iris it dwindles to almost negligible proportions. To compensate for this there is, however, a very thick adventitia of collagenous tissue, especially marked in the iris, almost exceeding in thickness the lumen of the vessels.

Dr. Swindle has a new conception or theory of presbyopia. To ophthalmologists it means a long-sightedness, especially that incident to old age, due to rigidity of the crystalline lens which renders accommodation difficult for near objects.

The present-day conception of the removal of the aqueous in the human by means of Schlemm's canal is amply confirmed. Before accepting a new idea such as proposed by the author, we should await further experimental evidence.

Dr. Peter C. Kronfeld said that in analyzing the vasomotor reactions to eserine, Dr. Swindle has stressed the very marked contraction of the arteries situated in the sclera and has considered the dilation of the arteries of the iris as a secondary phenomenon. Why does he assume that this is the actual relationship? Eserine applied locally, that is, to the surface of the eyeball, is bound to reach the vessels of the iris and of the sclera at about the same time. Further, the loose iris tissue should make it very easy for its vessels to dilate. It would seem more logical to assume that the dilation of the iris vessels was the primary phenomenon, especially since a vasodilator response to eserine is much more common and much better known than a vasoconstrictor reaction.

With regard to the association between glaucoma and shallowness of the anterior chamber to which the essayist referred in his concept of the mechanism of primary glaucoma, it has been shown statistically (B. Rosengren, Acta Ophthalmologica, 1930, v. 8, p. 99) that, leaving out the cases of acute uncompensated glaucoma, the anterior-chamber depth of glaucomatous eyes does not differ from that of nonglaucomatous eyes of individuals of the same age. In these cases of compensated glaucoma obviously the glaucoma is not characteristically associated with any particular depth of the chamber, nor does the glaucoma bring about any alteration of the chamber depth. In a series containing compensated and uncompensated forms of glaucoma, one finds, of course, a prevalence of shallow chambers. What the actual relationship between the shallow chamber and the occurrence of uncompensated glaucoma in these cases is, is not known with any degree of certainty. For a number of these cases it seems as though the shallowness of the chamber was essentially a nonglaucomatous feature which only predisposes the eye to glaucoma. In other types of glaucoma the shallowness of the chamber may actually be a glaucomatous feature; that is, an anatomic change brought about by or in the course of glaucoma. As to primary glaucoma in general, it does not seem to be correct to associate it with an unduly shallow chamber.

Dr. Louis Bothman remarked that the tension in the baboon's eye was given as

55 mm. Hg. Was that produced experimentally and, if so, how? Or did Dr. Swindle happen to get a rare case of an animal eye with glaucoma?

Dr. P. F. Swindle, in closing, referred to Dr. Theobald's suggestion that the injected India ink, instead of the eserine, might have caused constriction of the slender scleral arteries in some of the specimens shown. The importance of this question is clearly indicated by the observation that the skeletal muscles of a freshly killed animal react very strongly when India ink is injected into the arteries. One might presume, therefore, that the muscle fibers in the walls of the arteries must also react strongly, but he had no tangible evidence that the ink causes vascular reactions which prevent perfect injection of arteries anywhere in the body. Some of the best ink injections (of the kidney and eye, for instance) were obtained by injecting whole or diluted India ink into the arteries immediately after the animals were killed. The eye specimens in which the greatly constricted arteries of the circle of Hovius were seen, were injected with dilute, detoxified India ink immediately after the death of the animals. The injection pressure was low and pulsatile to avoid producing artifacts. Eserine was administered several minutes before the animals were killed. Cinnabar (in water) could not be urged, by injecting it rhythmically and under low pressure, into many of the small arteries of the circle of Hovius in the eserinized eyes, but it flowed readily into these vessels in noneserinized eyes and much more readily into the same vessels of the atropinized eyes. Properly detoxified India ink does not cause appreciable reactions of the skeletal muscles of a freshly killed animal.

It was emphasized in the slides that the circle of Hovius (commonly called the canal of Schlemm by Dr. Theobald and others) in the human eye is usually close to the drainage angle. Only a thin layer of scleral tissue may separate all or only a part of it from the angle at some points in both the human and the dog eye, while at other points in the same eyes the most anterior portions of the circle vary considerably in the human being as well as in other mammals.

In his effort to interpret his own findings in the light of some of Sondermann's statements, he had been forced to the conclusion that the intrascleral portions of the "inner canals" are some of his venae vestigia oculi, that these have open mouths at the scleral surface and that in late fetal life and in postnatal life they are not continuous with the intratrabecular spaces or channels which remained after the primitive intratrabecular blood vessels vanished. Upon following these spaces some of them lead one in the direction of some of the open mouths of the venae vestigia oculi, but also in various other directions and in such a manner that the pattern they form has an interesting resemblance to the original angioarchitectural pattern in this loose tissue.

Sondermann believes that his inner canals are vestiges of ontogenetically primitive vascular connections between the tunica vasculosa lentis. Collectively they are only an anterior portion of the deep scleral plexus. It is clear from the specimens that the anterior portion of the tunica vasculosa lentis is systemically connected with the anterior rim of the uveal tract by means of many anastomoses both before and after an iris can be identified as such, and that these anastomoses do not reach points either in the drainage angle or the sclera. As Dr. Goldenburg suggested, the thing of principal importance is the existence of the drainage channels, whether we choose to call them inner canals, venae vestigia oculi, or something else.

Dr. Kronfeld raised a question concerning the depth of the anterior chamber in glaucoma. It is true that the anterior chamber does not become shallower in some instances as glaucoma develops. He did not mean to imply that the petechial infarction is the primary etiologic factor in all cases, but it is a very important factor, usually the primary one in primary glaucoma and a secondary one in at least some cases of secondary glaucoma. Dr. Goldenburg suggested that buphthalmos is due to absence or relative absence of the venae vestigia oculi. If so, the anterior chamber should not be expected to become relatively shallow as the intraocular pressure increases.

He had long wondered about the nature of the mechanism of the eye which permits such a drug as eserine to get so readily from the conjunctiva to the sphincter iridis. In the vicinity of the limbus there are many little vessels that are close to the conjunctival surface onto which the eserine is dropped, and observations show that the blood often changes its direction of flow in some of these vessels. They are physiologically and pharmacologically inseparable from the vessels of the circle of Hovius. The eserine does not have to go far into the tissue to act upon some of the vessels that can cause the reaction of the iris. It might be expected that the eserine would reach the iris after a time. Probably the indirect effect of the eserine on the iris comes first, and eventually the drug reaches the sphincter iridis and thereby causes a further constriction of the pupil. Observations on eyes, such as the fox eye, indicated that there are really two mechanisms of accommodation, which can function together or separately. The human iris may fail to react to changes in light intensity and yet react with an effort to accommodation. Now and then the lens of the accommodated eye is so loose that gravity pulls it down and it often trembles when the eye moves. This cannot always be observed in a normal individual. It can be seen only when the vascular mechanism is working poorly or not at all. If the vascular mechanism works alone the lens is not loose, and if the two mechanisms work together the lens is not loose.

In reply to Dr. Bothman, the animal from which the glaucomatous eye specimens were obtained was a baboon, a mandril, and it had primary glaucoma. Glaucoma is rare in mammals, but it had been seen occasionally during the 16 years while he had acted as pathologist at the Washington Park Zoo in Milwaukee.

Robert Von der Heydt.

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TO DESIGNATE PRISM STRENGTH

The action of light on a prism differs essentially from the action of light on a lens. Lenses focus light. All the light coming from a certain luminous point and falling on a lens is brought to a focus in a single *point,* if it is a spherical lens, or a single *line,* if it is a cylindrical lens. The distance of that point, or line, from the lens indicates the strength of the lens: The stronger the lens, the quicker the rays are brought to a focus, the closer is the focus to the lens.

A prism does not focus light. All the rays from a luminous point that fall on a prism have their direction changed equally, whatever point of the prism they fall upon, so that there is no change in the relation to each other of the different rays coming from one point, but there is a change in the direction of the rays passing through all the points of the prism in exactly the same way and to the same extent. If the rays were parallel when they came to the prism, they remain parallel after leaving it, but in a slightly different direction. If they were diverging when they fell on the prism, they will diverge the same amount when they leave it, although moving in a different direction. If they were converging toward a point when they struck the prism, they will still be converging toward a point when they leave

the prism, but to a point in a different direction. The original point and the last one will be the same distance from the prism, but one will be in a different direction from the other.

The strength of a prism is measured by the difference in direction it makes in the rays passing through it. The strength of a prism is indicated by the angle of the change in direction of the rays that pass through it. It is the angle of the deviation the prism makes in the direction of the rays.

The strength of a prism varies with the angle at which the rays strike and leave the prism. The strength of a prism depends on the inclination of its two plane surfaces to each other. Formerly all prisms were designated by this angle. A 1-degree prism was one having its two plane surfaces meet at an angle of 1 degree. A 10-degree prism was one in which the surfaces met at an angle of 10 degrees. Two prisms having the same angle would have the same strength, if they were made of glass having the same index of refraction. But prisms made of different kinds of glass may have different strengths, although their surfaces meet at the same angle. Different substances have different effects on light passing through them. The effect of any transparent substances on light passing into it or from it is called its *index of refraction.*

Different samples of glass may have different indices of refraction. If the index of refraction for air is taken for 1.0 the index of what is called flint glass may vary from 1.57 to 1.70. The strength of a lens, or a prism, depends on both its shape and the substance of which it is made. The 1D. lens will bring parallel rays to a focus at one meter, whatever the index of the glass from which it is made. A 1-degree prism may change the direction of light passing through it 0.5 of a degree, if made of one kind of glass, and 0.7 of a degree, if made of another kind. If made of diamond it might change the direction of the rays 2.6 degrees.

For ophthalmic use, prisms should be numbered by their refracting power instead of by their shape. W. S. Dennett, in 1889, proposed a unit of refracting power for prisms to correspond to the numbering of lenses in diopters. With a glass commonly used for making spectacles, a prism having an angle of 1 degree between its refracting surfaces causes a deviation in the ray of about 0.57 of a degree. He suggested this should be the unit for prism strength, to be called a *centrad.* Prentice, the optician, suggested a nearly similar unit for prism strength, which he called a prism diopter. For weak prisms, such as are most frequently used in spectacles, there is not much difference between these two units. But for stronger prisms the centrad is mathematically correct, while the prism diopter is not. A 40-centrad prism has exactly 40 times as much effect on the rays passing through it as has 1 centrad. But a 40 "prism diopter" would have 42.29 times the effect of a 1 prism diopter.

In spectacle glasses, prismatic effects are often combined with lens effects. For this reason the best way to indicate the desired prismatic effect is often by the decentering of the lens. Through the optical center of the lens there is no prismatic effect; but rays passing through any other part of the lens are deflected, the deflection varying with the distance of the ray from the optical center. A table to show the amount of the decentering required to give a certain prismatic effect is convenient. Such a table was first published in the Transactions of the American Ophthalmological Society, in 1889. In prescribing to give a certain prismatic effect it is often best to designate the required decentering of

the lens in millimeters, with its direction, up or down, out or in. In rare cases it may be best to indicate the direction of decentering in degrees, as we would indicate the axis for a cylindrical lens.

Edward Jackson.

MOTIVATION AND VISUAL FACTORS

The small boy who steals is led to do so by some physical urge or necessity, or by circumstances of training or surroundings which have directed his mental processes into the appropriate channel. Likewise the industry of the almost blind man who wonderfully succeeds in earning a living in spite of his visual handicap is just as definitely the resultant of earlier factors as is the slothfulness of the visually capable individual who goes through life dependent upon public or private charity.

It is a fundamental principle of organized human society that each person of sound mind has the power to choose between good and evil and to base his acts upon that freedom of choice. Yet, since every effect has a cause, reason tells us that each "willing" results from antecedent conditions, either mental or material.

Much has been made of the influence of refractive errors in the shaping of character or career. Cases have been cited in which the previously inattentive and rebellious pupil was changed into a paragon of scholastic virtue upon being provided with a pair of glasses; although expectation of such miracles meets with many disappointments.

It does not require much experience or insight to realize that the personality of a child may be profoundly affected by such striking defects as squint or the disfigurement produced by an extensive corneal leucoma. It is almost equally obvious that the limitations imposed by a high myopia may restrict the contact of the individual with the external world and so direct him to pursuits which depend upon close use of the eyes.

In a general way it is probably true that the best refractionists are found among those who in their own persons have experienced decided inconvenience from refractive error. They who have had such personal experience and who have made a close study of refractive technique are likely to encounter patients in whose lives relatively moderate refractive errors have played an important part. No one who in his own person has been thus burdened would be apt to agree with the remarkable recent advice of a western ophthalmologist to the effect that glasses are to be prescribed only to improve vision!

The important part that a refractive error may play in the life of an individual is illustrated by the case of a lawyer who gave up the practice of his chosen profession because of inability to tolerate the necessary amount of reading. Direct and indirect evidence indicated clearly that he had never received an adequate correction for an important astigmatic error.

On the other hand, there are patients who display a remarkable tolerance for refractive errors which would prove disabling in the lives of others. There are also many inefficient pupils and workers who have no refractive error upon which to base an apologia, and whose inadequacy in the struggle of life must be attributed to defects of character or intelligence.

In the course of the past six years the Dartmouth Eye Institute has undertaken an investigation of the visual characteristics of the class of 1940 of Dartmouth College, with special reference to the influence of visual factors. The 124 mem-

bers of the class were studied by means of visual examinations, psychologic tests, interviews, autobiographies, and letters exchanged after graduation.

The faculty members whose names are upon the title page of the 369-page report of this investigation are: Irving E. Bender, Henry A. Imus, John W. M. Rothney, Camilla Kemple, and Mary R. England. With them coöperated, among others, Drs. W. B. Lancaster, Prof. Adelbert Ames, the late Dr. A. Bielschowsky, and Dr. H. M. Burian. (Motivation and visual factors, Dartmouth College Publications, 1942.)

The Dartmouth report, which represents an enormous amount of work faithfully performed, consists chiefly of a study in the developmental history of a series of college students. The lives and characters of twenty selected cases are related at rather considerable length.

The analysis of "visual factors" assumes a somewhat insignificant place in "motivation" as here described. At times one even wonders how much practical benefit is to be expected from such an inquiry, and whether it achieves much more than the elaboration of a typical "problem" in psychic study, assigned by a college leader to his associates and pupils. The report is occasionally burdened with a sort of psychologic "lingo."

Inevitably, a few of the students are precociously sophisticated, while others sound a bit simple in their outlook. There is the youth who is "slouchy in his movements and loose-jointed in his gait," whose "frequent laughs and grimaces, and sweeping gestures, give him a happy-go-lucky appearance." "High in his scores in the reading tests and moderately high in the A.C.E. Psychological Examination, Edward did not live up to academic expectations." He "derives from old Colonial stock. His immediate family . . . have acquired considerable wealth." "The paternal domination seems to have resulted in Edward's passivity." His ocular defects caused him no visual difficulty.

We read of "Alvin," who combined stern self-discipline with emotional asceticism, who since boyhood had taken a profound interest in trains and railroads, and who from the tradition of family unity and loyalty through generations seemed to derive a sense of security in a "crazy world." For him the use of glasses represented a concession to weakness.

Then there is "Bill," who maintains the spontaneous egocentricity of a child, who according to one commentator "is characterized by his beaming face and enthusiastic attitude, which, I believe is often artificial," whose early childhood was spent with his grandmother, but whose emotional relations to his parents were arid, although he has a vivid Einfühling into his mother's troubles. He posseses an "inordinate desire for attention and affection," and values his glasses because he thinks they may have been responsible for his overcoming a slight stammering.

"Jimmie," for whom "life is a large pond in which he is a small frog determined to become bigger," is preoccupied with his self-analysis and a tendency to emphasize his hostile attitudes and intentions toward the world." He considers aniseikonic lenses unusually disfiguring, but, had they cost no more than ordinary glasses, he might have accepted them and found them helpful. He would hardly say, however, that his lower marks were caused by his eyes.

The student whose pseudonym is "Temple" has been accused of arousing animosity "through apparent egotism, emphasis on rationality and strength of will, the 'monotone' of his personality, the overseriousness, the lack of humor."

His record points to overprotection by the mother, resulting in overdependence and a feeling of inferiority. He gets high grades and rejects a fuller correction of his hyperopia.

"Jonathan" shoots worse golf when playing with his father than when with others. He has an eagerness for dangerous exploits, but his grades fall off after glasses are prescribed.

"Philip," who came from a broken home, frequently stole from his father during childhood but becomes "one of the most stable and reliable boys in school" and earns the entire confidence of his fellow-students and the faculty. He discards his half-diopter minus cylindrical lenses axis ninety when advised that they are unnecessary, and reports later that he has suffered no visual discomfort.

The visual factors mentioned in relation to the twenty sample cases include the various combinations of hyperopia, myopia, astigmatism, anisometropia, aniseikonia, strabismus, and other muscle anomalies. The suggestion is offered that the assembled information is not such as to make it possible to draw conclusions "regarding the influence of visual defects on the motivational pattern of the individual. . . . The motivational pattern . . . does influence his adjustment to his visual condition. In every case, the visual condition is so deeply embedded within the total personality that its psychological significance is not clear."

One feels a little doubt whether "Dartmouth College Publications" has sufficiently considered its statement (on the cover of the volume) that "this report, awaited eagerly by eye-specialists, . . . throws light on the important relation between motivational and visual factors at the college level."

Some ophthalmologists will hesitate to agree with certain individual assumptions concerning refraction. A student with alternating convergent strabismus had a greater hyperopia in the right than in the (dominant) left eye, but was ordered a higher correction for the left eye "in order to blur the left dominant eye," a size difference of 0.75 being added. It is hardly surprising that he ceased using the glasses because they blurred vision and did not cure headaches; or that his discomfort was later relieved by a new prescription from his own ophthalmologist which added the correction of a slight amount of astigmatism.

Is it certain that the student whose total error called for plus 0.62 sphere in the right and plus 0.37 sphere in the left eye, with a low minus cylinder equal for both eyes, "needed no visual correction"? Or was it proper to give no prescription to the student who called for Right plus 1.25 sphere minus 0.37 cylinder axis 10 degrees, Left plus 1.25 sphere minus 0.37 cylinder axis 180 degrees, who made no complaints but who had ineffective work habits derived from feelings of inferiority and fears of independent judgment? Perhaps the visual factor was thought of as being by this time "embedded in the total personality!"

Is 1.50D. a small amount of myopic astigmatism, as suggested in the case of "Arthur Robins"; and is it proper to say that the eye in question, the former cylindrical correction of which had been 0.50D., had been "slightly undercorrected"? It would not be difficult to refute the statement that low myopic errors do not produce eyestrain; or the related statement that full correction of a myopic error necessarily causes more ocular strain than a partial correction.

In any report from the Dartmouth Eye Institute, aniseikonia naturally plays a prominent part. On this score the sample case histories are about in line with gen-

eral experience.* Some student patients thought the aniseikonic lenses very unsightly. Others felt better without than with the aniseikonic correction. A few felt distinct benefit from correction of the size difference. One student is frankly described as "endeavoring to persuade himself that the glasses did relieve his visual fatigue." A number of low aniseikonic defects are interpreted as due to difference between the refractive corrections of the two eyes.

It will occur to many readers of the Dartmouth volume that the influence of visual factors is studied here in relation only to a single rather lengthy episode in the life of the individual. The life of the subject would need to be followed from the cradle onward, with due consideration of the mass of detail available from parental and other personal contacts and in regard to other environmental circumstances.

The task approaches the impossible. The theoretically perfect analytical parental mind, with preconceived and unvarying purpose, following closely the life of the offspring, might be capable of reaching fairly reliable conclusions, provided the visual problem were sufficiently pronounced and instructive. But such a combination is unavailable. As with so many other human problems, the unknowns outweigh the knowns.

W. H. Crisp.

HIDDEN FEES

After a week spent in Atlantic City in frequent talks with many of the ophthalmologists gathered there, two problems appeared to stand out as demanding prompt consideration. The first one, that of ophthalmo-optometric relations, was discussed by the writer in an editorial in the May issue of this Journal. At the recent meeting the eye section reversed its action of June, 1941, when it voted to rescind the ruling of 1935, which prohibited physicians from certain relationships with nonmedical persons, such as the teaching of optometrists by ophthalmologists. As the rule now stands, this is still prohibited. The merits of this particular action will not be discussed here, nor will any part of this controversy be considered except for the mere statement that it would seem a pity to close the door on further conferences on the matter. The writer's editorial on the subject suggests a possible method for discussion of the problems common to ophthalmologists and optometrists.

But the subject of this editorial is that equally difficult one of rebates, kickbacks, and the like. There is a growing dissatisfaction in the Government and better-business bureaus with the matter as it now stands, and many ophthalmologists also are unhappy about it.

The question as it concerns the Government and as it concerns the medical profession is somewhat different. The Government has interested itself in the welfare of the common man and will not much longer tolerate his being subjected to secret charges or his having to pay a hidden premium to a refractionist. The profession, on the other hand, further than being interested in welfare is concerned with the ethics of this subject. Let us consider why our code of ethics (which most of us, I am sure, think is fundamentally sound) is so often grossly

* Says Field (Jour. Amer. Med. Assoc., 1942, v. 119, May 9, 132) ". . . the majority of patients who are referred for aniseikonic measurements are in need of (1) a more adequate refraction and (2) an investigation of binocular vision from the point of view of muscle balance, vergence powers and stereopsis. The vast majority of patients will be relieved of asthenopic symptoms if the needs mentioned are first properly attended to. After that has taken place, aniseikonia should be considered."

flouted. The primary reason is that receiving remuneration from prescribing and furnishing glasses, in addition to that from the actual testing of the eyes, is so tempting that a majority of ophthalmologists are unwilling to forego it. There is certainly a justification for an additional service charge being made by those who actually handle the fitting of the frames, and conduct the business of the transaction; but more of this later. In the second place, the minority who accept no remuneration other than for the refraction do not wish to make an issue of this matter because they do not feel "holier than thou" and certainly do not want others to think that they entertain such an attitude. Furthermore, it would be necessary to bring accusations against some individual or group among whom would be found their very good friends. They may think that probably in many cases the circumstances justify the actions. What, then, should be done? Rather obviously action in the matter is going to come from without if not from within. It will be much better for the public and for ophthalmologists if from within.

It is obvious, as mentioned earlier, that some ophthalmologists do give a service beyond refraction and should be paid for this. Some assume a financial risk; for example, the possibility that glasses which have been ordered by the patient may never be paid for, breakage, and other costs. Ophthalmologists must have some compensation for these losses. It may be desirable to pass all of this grief on to someone else, as may those who practice in communities where there are prescription opticians. Speaking as one such ophthalmologist, I regard it as great good fortune to be so located that the necessity of entering into the optical business is not upon me. There is no virtue in this attitude, but a lot of expediency!

After this preamble I make bold to suggest a plan—not as a novelty, because there is certainly nothing new in the idea, but rather because I entertain the belief after long talks with judicial committees, ophthalmologists, retail and wholesale opticians, that some such plan, if presented now, might overcome the most serious objections and if elaborated a bit might be acceptable to all concerned; except to the ophthalmologist who wants something for nothing.

The fundamental principles are two: First, there must be nothing hidden in the transaction between ophthalmologist and patient; second, there must be no remuneration to the doctor except for a service rendered or something material, such as glasses, sold to the patient. The payment of a "kickback" to the ophthalmologists by a wholesaler, or by anyone else who supplies the glasses to the patient and who renders the service of fitting and later care, has no possible justification in honor and fairness and cannot be tolerated in any upright program; for one reason because in the final analysis this cost must be passed on to the patient. That kind of thing will have to go. That many practice it is no secret, and those who do are known to the Government because this information is required by the Government from wholesalers and retailers for income-tax purposes, and we can leave the Government to deal with these ophthalmologists, as it surely will, because as much as this has already been said by its agents. If anyone can afford to "kick back," he is charging too much, and his charges should be reduced. Ophthalmologists can patronize those who are able to charge less because they do not have to meet this additional expense, and if there is any combination to restrain a firm from charging less than its competitor, then it *is indeed* a matter for the Government

to handle! It is greatly to be hoped that the names of those who accept "kickbacks" are never exposed, because embarrassment between erstwhile friends would inevitably follow. Rumor hath it that in any large center ophthalmologists are listed by lens dispensers as in groups A, B, or C. Those in Class A never accept "kickbacks," those in B only occassionally and very secretly, and those in C accept them openly.

Most optical wholesalers represent a high type of business man. Is it not possible that they would coöperate with ophthalmologists by refusing to pay percentages to referring ophthalmologists merely for referring the patient? Surely it would be economically advantageous to them.

Probably most kickbacks are from retailers, and they cannot easily be reached. But, if the American Medical Association will take a definite stand and will confer with the only authority qualified by having both the information and the power to act and willing to coöperate, namely the Government, the whole matter can be cleared up!

Leaving out of consideration the group that wants something for nothing, because we could not justify this practice of theirs even if we would, the suggestion is tendered that those who merely perform refractions bill the patient for this only, as heretofore; that those who measure for frames and furnish glasses charge the patient for the refraction, and for the actual cost of the glasses as charged by the wholesaler, and add a service charge which will cover their time and overhead; and that they list these three items separately on the bill given the patient. This would eliminate any hidden charges, would leave the patient in no doubt as to the cost of the business transaction, and in the writer's opinion would be entirely ethical. It would exceed any governmental requirement, because there is no law that a purchaser be informed of the cost price of the article to the seller. But to surpass the law in this would certainly not be open to criticism.

This subject will not down. It is almost certain to be brought up at the next session of the American Medical Association. Perhaps this Journal would make a good clearing house for a preliminary discussion; it is even possible that some plan acceptable to the large majority of ophthalmologists might be worked out and could be presented to the Section and subsequently to the House of Delegates for action.

The writer is well aware that the ophthalmologist is not the only offender on kickbacks, and it may reasonably be asked why he should be the scapegoat. Percentages are allegedly paid on other appliances ordered by physicians and sums paid for refers to laymen in businesses from which such reference can profit. But let us not ask, "Why pick on us?" but accept the problem that is definitely ours and try to solve it.

Lawrence T. Post.

THE INTERAMERICAN MEETING OF THE AMERICAN MEDICAL ASSOCIATION

It was fitting that to the greatest and most representative of the American medical societies fell the task of initiating and cultivating friendly relations with the physicians in all the countries of Latin America. For some years several Sections of the A.M.A. have been inviting individual guests from these countries, but lately the Association as a whole has sent invitations to prominent physicians to attend the meetings and also to contribute to the scientific exhibits. The war

in forming closer bonds between all countries of America has also encouraged a more friendly intercourse between its physicians.

At the last meeting of the American Medical Association, in Atlantic City, from June 8th to 12th, a large number of Latin-American physicians participated in the programs of the Sections. One hundred and seventeen of them came from almost all of the countries of this part of the hemisphere, the largest number, we believe, ever to have assembled for the meetings of the A.M.A., and surprisingly large in view of the serious disruption of transportation coincident with the war. The majority of these physicians made the trip by airplane, some with great difficulty and at great expense. It is to the credit of the Association that they found the meetings and exhibits so interesting and profitable for learning, that they felt compensated for the hardship and danger entailed in the trip. With European clinics and societies closed because of the war, it was natural that all progressive medical men in Latin America should endeavor to follow the advance of the medical sciences in the United States.

The meeting in Atlantic City was for some of them a revelation of the great progress, not only clinical but especially in the field of research, that has been achieved in this country. They were full of praise for the institutions which had furthered investigation in, and the practice of, medicine. Some of them said they foresaw the day when medicine in this hemisphere will be far in the lead over medical research in Europe. However, to realize this objective it is necessary not only that they know the work we do here, but it is imperative that we know and appreciate more fully what is being done in progressive medicine in all countries of Latin America beginning with Mexico and down to Argentina and Chile.

The majority of the Latin-American physicians who came to the American Medical Association meeting were of high standing in their respective countries, and we could certainly learn from them in many respects. Medicine in some of their countries has had a remarkable history. At the end of the eighteenth and beginning of the nineteenth century there were in Mexico and Peru excellent physicians who had been educated in Spain and were practicing medicine and surgery, and good results. Later, when those countries became independent, numerous students went to Europe, especially to France, and absorbed all that was known of medicine and surgery in their day. At the end of the last century, men like Vertiz, Liceaga, and Carmona in Mexico, Razzeti in Venezuela, and others were excellent clinicians in the best traditions of the French and German schools. The development of modern medicine in those countries, however, was handicapped by unstable political conditions, by lack of laboratories and resources for medical research and travel. The development of medicine in Argentina, Brazil, and Chile is more recent, but its growth has been faster than in the more northern countries, due to more stable political conditions and the acquisition of national wealth, which has been reflected in the erection of modern hospitals and laboratories, and in the provision of better medical education and facilities for research and travel.

Ophthalmology has made rapid advances in Latin America, and the contributions of men in Argentina, Brazil, Mexico, Cuba, and elsewhere are in some respects outstanding. It would be to our advantage to promote a closer relationship through Pan-American Congresses and medical journals. The greatest barrier until now has been the difference in language, but we know of some progres-

sive physicians in the United States who are studying Spanish now and soon will be proficient enough to understand and speak the language. It would be consistent if our essayists were to include in their bibliographies references to the contributions in Spanish and Portuguese by our Latin-American colleagues.

Manuel Uribe Troncoso.

RESEARCH IN OPHTHALMOLOGY

The thirteenth annual meeting of the Association for Research in Ophthalmology was an extremely interesting one. In addition to the excellence of the papers presented, the effect of the emigré on the future of research in ophthalmology in America was apparent.

The European influence on ophthalmology has been felt before. Until relatively recently the majority of the leading oculists were foreign born and trained or had had at least a part of their training abroad. This served as a stimulant to ophthalmology as a whole and ended, partly through the efforts of the American Board of Ophthalmology, in the establishment of postgraduate training in the United States to the point where the American-trained ophthalmologist is comparable to any in the world.

With the development of postgraduate training, it was only natural that certain teaching centers should be established and that these centers should offer facilities and encouragement to the younger man interested in research. The result has been that during the last 10 years an increasing amount of very excellent research work has been done in various institutions, especially in the fields of bacteriology, pathology, physiology, and biologic chemistry. The practical application of much of this laboratory work to clinical ophthalmology has already been made. In addition, the results of laboratory research have received sufficient recognition that funds have been made available to enlarge the scope of the work carried on. The public has also manifested a greater tendency to make gifts and endowments for ophthalmology.

Until recently Europe was considered the center of research in ophthalmology. Thus it was necessary for the well-informed ophthalmologist to be conversant with the foreign journals. With the outbreak of war there was an influx into this country of foreign ophthalmologists. Among them was a certain number of relatively younger men with excellent training in research. These have found a place in various institutions where they have continued their work and where they have further developed an interest in research.

Another factor instrumental in the recognizance of research has been The Association for Research in Ophthalmology. At the meetings of this society it is possible to present in detail work that often is not acceptable on a clinical program. At the earlier meetings of the society, the program was often primarily a clinical one and included such subjects as a symposium on acute iritis or chronic uveitis. The number of clinical papers has gradually decreased until at the meeting this year eight papers were presented, all of them based on extensive research.

A review of the localities from which these papers came shows the widespread interest in the subject. The Mayo Clinic, The Presbyterian Hospital of New York, The State University of Iowa, Massachusetts Eye and Ear Infirmary, Illinois Eye and Ear Infirmary, Wayne University of Detroit, College of Physicians and Surgeons, Columbia University, and the University of Cincinnati were represented on the program. In addition, there are

many other institutions where similar research is being conducted.

It was of interest that three of the papers were on the aqueous and anterior chamber and represented laboratory work that ultimately may have a direct bearing on the clinical treatment of glaucoma.

Manuel U. Troncoso's paper was on the intrascleral vascular plexus and its relation to the aqueous outflow; Peter C. Kronfeld, H. Isabelle McGarry, and Homer E. Smith discussed gonioscopic studies of the canal of Schlemm, and K. W. Ascher's dissertation was on the aqueous veins in relation to the physiologic importance of the visible elimination of the intraocular fluid. Robison D. Harley's work was a quantitative study of the cells and fibers in the nucleus-nerve complexes of the fourth and sixth cranial nerves, while Kenneth C. Swan and Norman C. White presented a discussion of the corneal permeability in relation to the factors affecting the penetration of drugs into the cornea. T. L. Terry, who has done a good deal of research on the fibroblastic overgrowth of persistent tunica vasculosa lentis in infants born prematurely, gave a paper on studies in the development and involution of the hyaloid artery and tunica vasculosa lentis.

Erich Sachs presented his observations on the physiology of the ciliary muscle as based upon some extremely ingenious experimental studies. Ludwig von Sallmann's paper was on sulfadiazine iontophoresis in pyocyaneous infection of rabbit cornea.

A review of the program reveals that three of the eight papers were given by recent emigrés, three were presented by native-born citizens, and the remaining two by foreigners who have been on our shores for a longer period of time. As to the quality of the work done there was no choice among the various groups.

Perhaps one reason why the foreign research worker in ophthalmology has been able to contribute so much in the way of experimental work, much of which is applicable to the clinic, was his freedom from the cares of financial responsibility and the demands of a private practice. Many of the emigrés have come from clinics subsidized by the state, where appointments were made on research ability, based on a thorough training. The American ophthalmologist, on the other hand, has been handicapped in the past by the relatively few opportunities to engage in pure research unhampered by private practice and the need for earning a livelihood. The development of large laboratory centers in ophthalmology within comparatively recent years has done much to supply this need. The emigré appears on our shores at a time favorable to him and to us.

So much excellent work has been done in this country that research in ophthalmology is firmly entrenched. The work of our own men further stimulated and aided by the work of the recent emigré can leave little doubt as to the glorious future of research in ophthalmology in this country. Frederick C. Cordes.

BOOK NOTICES

NEW MANIFESTATIONS OF INTERNAL DISEASES. By I. S. Tassman, M.D. First edition. Clothbound, 542 pages, 201 illustrations, including 19 in color. St. Louis, 1942, Press of The C. V. Mosby Company. Price $8.50.

No outstanding book of this character has been published in America recently. The best book heretofore has been that by Foster Moore, which is now 10 years old, so this addition to our literature is particularly desirable. The first 6 chapters of 245 pages are devoted to a description of the normal and abnormal

structures of the eye. The inclusion of this material seems perhaps a little dubious as being not entirely related to the subject; but it is well done and probably is justifiable. The remainder of the book, practically two thirds, has to do with congenital and hereditary eye manifestations; infections and infectious diseases; tuberculosis; virus infections; fungus infections; ocular parasites and parasitic infections; focal infections; drug and chemical intoxications; diseases of the cardiovascular system; diseases of the blood; disorders of menstruation and pregnancy; diseases of the endocrine glands and metabolism; avitaminosis and diseases of nutrition; diseases of the nervous system; intracranial tumors; diseases of the skin; diseases of bones of the skull. Obviously this is a rather complete presentation dealing with systemic lesions. In each case a short description of the general pathology and clinical picture is given first, and this is followed by the ocular manifestations.

Most of the illustrations are very good, being particularly adapted to demonstrate the points in question. I was pleased to find a reference to a rather obscure condition that I had been unable to locate in any of the modern textbooks of ophthalmology. The book would appear to be excellent for the teaching of the student, who should be impressed particularly with the relations of ophthalmic diseases to general medical conditions. It is nicely prepared with good paper and good binding, as are all the Mosby products. I regard it as a valuable addition to one's ophthalmic library.

Lawrence T. Post.

MOTIVATION AND VISUAL FACTORS: INDIVIDUAL STUDIES OF COLLEGE STUDENTS. By Irving E. Bender, Henry A. Imus, John W. M. Rothney, Camilla Kemple, and Mary R. England. Clothbound, 369 pages. Hanover, New Hampshire, Dartmouth College Publications, 1941. Price $4.00.

(See editorial, this issue, page 998.)

TRANSACTIONS OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY. Volume XXXIX, 1941. Clothbound, 565 pages. Philadelphia, William F. Fell Co. Printers.

The seventy-seventh annual meeting of the American Ophthalmological Society was held at Hot Springs, Virginia, May 29, 30, and 31, 1941. Twenty-two scientific papers were presented during the meeting.

Haden presented a series of lantern slides demonstrating the generally accepted theories of the embryonic formation of the vitreous.

Adler and McDonald, after studies on the pigmentary degeneration of the retina and the role of the melanophore hormone of the pituitary gland, concluded that in rats and frogs there was no appreciable effect on the retinal pigment following injections of the hormone. The assay of the blood of patients suffering from retinitis pigmentosa for a melanophore-expanding substance is not of diagnostic value.

Samuels presented a study of the lesions of the lens caused by purulent corneal ulcers. In severe conditions necrosis of the subcapsular epithelium occurred; in milder cases the subcapsular epithelium proliferated.

Clapp found atrophy of the capsular epithelial cells in 70 cases of immature cataracts removed in capsule and concluded that these changes were the cause rather than the result of the lenticular changes.

Arnold Knapp advises the operative treatment of congenital subluxation of the lens if satisfactory vision cannot be obtained by refractive means. He also reported two cases of retinal periphlebitis in the course of acute exudative choroiditis.

Gradle and Sugar report wound rupture after cataract in 21.5 percent of 200 consecutive private cases. They conclude that forcible contraction of the orbicularis and/or, of the ocular muscles may increase the intraocular pressure and rupture the wound.

Beethan employed iridodialysis in 122 operations for cataract extraction; the Verhoeff intracapsular technique was followed. The results were excellent.

Kirby's paper "Procedures in intracapsular cataract extraction," is a discussion of the various methods of intracapsular extraction with a clear description of the author's methods of applying pressure, traction, rupturing the zonule, and delivering the lens. The methods of stripping the resistant zonular membrane from the superior equator of the lens is original and has been practiced in difficult cases.

Jackson reports a series of 61 cases, followed an average of four-and-one-half years after cataract extraction—14 patients of the series were followed over 10 years. He has used the corneal section without sutures with success. A period of six months following extraction should elapse before glasses for near vision are prescribed.

Reese and Calhoun report five cases of rhabdomyosarcoma of the orbit; 14 additional cases were collected from the literature.

Schoenberg's survey of the records of glaucoma patients from three large clinics proves the inadequacy of the care of patients with glaucoma. The very small percentage remaining under observation indicates the general lack of treatment.

O'Brien and Swan have used doryl, a synthetic choline product, combined with a surface-tension-reducing substance—that is, zephiran—in the treatment of glaucoma. The combination advocated is a powerful miotic, reduces intraocular pressure, and produces a spasm of accommodation.

Dunphy and Albaugh report favorably their experiences with cyclodiathermy in the treatment of glaucoma. Their technique of nonperforating diathermy is simple and avoids the usual complications of intraocular surgery.

MacDonald discusses the ocular kinetic righting reflexes and describes a new instrument, the clinometer, which is used in testing. Judgment, training, and ocular dominance play an important part in acquiring the righting reflexes.

Verhoeff reports his personal experience with Moore's "lightning streaks." He concludes that the streaks are dependent upon the separation of the vitreous from the retina.

Benedict discusses the causes of blepharospasm. Treatment by injecting 2-percent novocaine, followed by 1 c.c. of 95-percent alcohol into the parotid plexus has been satisfactory.

Bedell reports six cases of papilledema without increased intracranial pressure. The cases prove the value of colored stereoscopic fundus photographs.

Gifford's paper on ring ulcer of the cornea emphasizes the need of an early application of a sterilizing agent to the cornea. The careful application of pure trichloracetic acid to each infiltrate is of value. In the presence of a well-developed ring infiltrate decompression by a limbal paracentesis or a form of keratotomy is indicated.

Cowan and Klauder record 12 cases of ocular pemphigus; the associated skin and mucous-membrane lesions have been

correlated. The services of a competent dermatologist are essential.

Berens, Rothbard, and Angevine report numerous cultural studies on patients with uveitis. An occasional blood culture of Staphylococcus albus was obtained. Positive intracutaneous reactions for undulant fever were obtained in 3 of 56 patients; one had an agglutinin titer of 1:40.

Lewis used sulfanilamide, sulfapyridine, and sulfathiazole in the treatment of 120 cases of gonococcal conjunctivitis. The early adequate systemic treatment with sulfapyridine is advised.

De Long and Howland report new aspects of ocular microörganisms revealed by the electron microscope. Pneumococci, streptococci, staphylococci, tubercle bacilli, and B. xerosis were studied under a 10,000 to 16,000 magnification.

A section of 215 pages is devoted to theses for membership in the society. The thesis of Raymond L. Pfeiffer on the roentgen ray in ophthalmology deserves careful study by all ophthalmologists.

This volume is an excellent contribution to medical literature and sets a high standard in scientific work.

William M. James.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

1

GENERAL METHODS OF DIAGNOSIS

Friedman, B., and Keil, F. C., Jr. **The Vollmer tuberculin patch test.** Arch. of Ophth., 1942, v. 27, April, pp. 728-732.

Eighty-eight patients from various ophthalmic services at the New York Eye and Ear Infirmary were tested with the patch test on one arm and a Mantoux test on the opposite arm. Of these, 71 were positive reactors and 17 negative reactors. The reaction to the Vollmer patch test was positive in 62 and the reaction to the Mantoux test was positive in 70. Of the 26 with the negative Vollmer patch test, 10 were found to be sensitive by the Mantoux test, whereas only one patient with a negative Mantoux test had a positive patch test. Generally the reaction to the patch and the reaction to the intracutaneous test were parallel. Probably because the group was preponderantly adult, the youngest being 11 years of age, the rating for the Vollmer test was lower in this series. The rating is sufficiently high to warrant advocating the Vollmer patch test as a preliminary step for determining sensitivity to tuberculin as it provides the desired information with a minimum of objectionable features. In the event of a negative reaction, a Mantoux test could then be made. In this series there were no necrotizing reactions and only a few delayed reactions. J. Hewitt Judd.

Kauffman, M. L. **Protection for spectacle lenses during ophthalmoscopy.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 589-590. (One illustration.)

Löwenstein, O., and Friedman, E. D. **Pupillographic studies. 1. Present state of pupillography; its method and diagnostic significance.** Arch. of Ophth., 1942, v. 27, May, pp. 969-993.

The authors describe their method of recording pupillary movements by photography, which permits exact measurement of the movements. Their findings are presented under the following topics: reflex to light, phenomena of fatigue, psychosensory-restitution phenomenon, reflex to darkness, reflex to near and to distant vision, the Westphal-Piltz (orbicularis)

phenomenon, tonohaptic reaction, reaction to light "by steps" (in cogwheel fashion), the analogy between physiologic-fatigue phenomena and pathologic-disintegration phenomena, asymmetric fatigue symptoms, psychosensory-restitution phenomenon in association with organic lesions and anisocoria; and convergence and divergence in the pupillographic picture. The findings are presented in graphs. Pupillography offers considerable aid both in general research and in the diagnosis of early lesions of the central nervous system. J. Hewitt Judd.

Re, B. V., Picoli, H. R., and Tettamanti, J. **Headaches of ocular origin.** Arch. de Oft. de Buenos Aires, 1941, v. 16, March, p. 109; April, p. 163; May, p. 225.

The authors describe in detail the different types of headache of ocular origin, and discuss at length their diagnostic significance.

Plinio Montalván.

2

THERAPEUTICS AND OPERATIONS

Connell, E. S., and Trowbridge, B. C. **Local use of sulfanilamide and derivatives in practice of ophthalmology.** Arch. of Ophth., 1942, v. 27, April, pp. 705-706.

Favorable clinical results have been obtained in lacerated wounds of the eyelid, streptococcic conjunctivitis, streptococcic ulceration, and trachoma with sulfanilamide powder. Sulfapyridine gave satisfactory healing of pneumococcic conjunctivitis and ulcus serpens, but caused more discomfort than either sulfanilamide or sulfathiazole. A 25-percent concentration of sulfathiazole in an oxycholesterol-petrolatum-ointment base is recommended for perifolliculitis, blepharitis, squamosa, and blepharitis ulcerosa, but for a pneumococcic and staphylococcic infection of the conjunctiva and the cornea, the powdered form is better. Since local application does not materially affect the level in the blood, it may be safely combined with oral or intravenous administration.

J. Hewitt Judd.

Grolman, G. von, and Angel, E. **Ophthalmologic study of drugs acting upon the peripheral circulation. 2. Local acetylcholine.** Arch. de Oft. de Buenos Aires, 1941, v. 16, June, p. 283.

The author has studied the action of acetylcholine upon the ocular circulation in thirty patients suffering from different forms of retinal and optic-nerve disease, including glaucoma. Both retrobulbar and subconjunctival injections of the drug were employed.

The retrobulbar injection was well tolerated and was followed by slight pain, congestion of the superficial blood vessels of the lid and conjunctiva, and moderate edema of the orbit and frequently of the eyelids. There was an increase in intraocular tension and in the blood pressure in the central retinal artery. Pupillary changes were absent. The balance between retinal blood pressure and intraocular tension leans toward the former and it is to be expected that the blood irrigation will be increased. In glaucomatous eyes in which the injection was given in an attempt to control or reduce central field defects, there were no symptoms of decompensation despite the rise in intraocular pressure. Quantitative campimetric changes were observed after retrobulbar injection of acetylcholine.

The authors believe that retrobulbar injection of acetylcholine repeated at frequent intervals is the method of

choice in the treatment of acute closure of the central retinal artery. Subconjunctival injection of acetylcholine has a somewhat different action. It produces miosis and a reduction of intraocular pressure in glaucoma, but the tension of normal eyes remains unchanged. (Tables, visual-field charts, photographs, bibliography.)

Plino Montalván.

Post, M. H., and Moor, W. **The sterilization of sharp instruments by chemical solutions.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 579-584. (6 exhibits, 2 references.)

Scheie, H. G., and Leopold, I. H. **Penetration of sulfathiazole into the eye.** Further studies. Arch. of Ophth., 1942, v. 27, May, pp. 997-1004.

A series of experiments are reported in which a microcrystalline form of sulfathiazole was administered by the intraperitoneal route. Preliminary studies showed that excellent levels of sulfathiazole in the blood resulted and that penetration into the aqueous of normal eyes was comparable to that in previous studies with sodium sulfathiazole given orally. Specimens of aqueous from eyes with experimental infections showed sulfathiazole levels proportionate to the severity of the inflammatory reactions. Eyes with fulminating experimental abscesses of the vitreous yielded aqueous containing 91 percent as much sulfathiazole as the blood. The authors found that the sulfathiazole content of the aqueous could be increased by vasodilator drugs, such as mecholyl hydrochloride and histamine. The authors conclude that until further evidence is accumulated, the use of this drug in cases of ocular infections is probably not contraindicated. J. Hewitt Judd.

Schupfer, Francesco. **A possible increased action of acetylcholine with ascorbic acid in the ophthalmologic field.** Boll. d'Ocul., 1940, v. 19, April, pp. 323-332.

The results (given in tabulated form) of Schupfer's test on enucleated frog eyes show that ascorbic acid increases the miotic action of acetylcholine only if the pupil has been dilated (by adrenaline), that both ascorbic acid and acetylcholine have a miotic action on the normal-sized pupil but that these drugs show an antagonistic effect if acting simultaneously, that is, their miotic action is not demonstrable. Tests made on the isolated iris of the ox show that ascorbic acid increases the miotic action of acetylcholine. These experiments indicate, among other facts, that one substance (such as ascorbic acid) may increase the action of two other drugs that have opposite actions (acetylcholine, in this instance, acting on the parasympathetic and adrenaline on the sympathetic). Endeavoring to explain how the two drugs exerted their miotic effect on a pupil dilated by adrenaline, the writer suggests that it is much easier to relax a muscle under strain than a muscle in the tonus of rest. (2 figures.)

M. Lombardo.

3

PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Brobeck, V. H. **Aniseikonia.** Rocky Mountain Med. Jour., 1942, v. 39, Jan., p. 38.

The article deals with the fundamental principles of aniseikonia. The author, who is director of the Aniseikonia Clinic at Colorado Springs, concludes that it is reasonable to assume now that of 4,000 people who

have been measured for aniseikonia, relief has been obtained from symptoms such as physical, emotional, and psychological disturbances in 65 to 75 percent. A 1.5-percent difference in image size is sufficient to cause trouble, but relief has been obtained by correction of less than this amount.

Ralph W. Danielson.

Crozier, W. J., and Wolf, E. **Theory and measurement of visual mechanisms. 8. The form of the flicker contour.** Jour. Gen. Physiology, 1942, v. 25, Jan. 20, p. 369.

This article contains a discussion of and several graphs showing flicker-response curves. F. M. Crage.

Di Bari, Enzio. **Vision through inclined prisms.** Boll. d'Ocul., 1940, v. 19, May, pp. 390-406.

By logarithmic-trigonometric calculation, the writer determines the astigmatism of thin prisms at different angles of incidence and varying distance of the object. He determines also the tolerance to the prism inclinations and deduces some valuable rules for ocular practice. (Bibliography, 8 figures.)

M. Lombardo.

Goebel, O. **Perception of whiteness. Excitation of the retina by longitudinal radiations?** Graefe's Arch., 1940, v. 142, pt. 4, pp. 491-494.

Previously the author has suggested that in the cones which perceive whiteness there are resonant oscillations of various frequencies, as is the case for the cones which perceive colors. As evidence for the resonance theory is the fact that the sensation does not cease with interruption of the stimulus but continues as an after-image. There is another type of light perception which produces only slight after-images, and at low illuminations, at least, produces intermittent excitation. The author speculates about these two types of perception in the light of current physical theory, and suggests that the first type, perceiving color and whiteness, is caused by excitation of the cones responding specifically to transverse oscillations of light; while the second type, perceiving glare, (Glanz, Glühen, Leuchten) arises from stimulation of the rods, which respond specifically to longitudinal oscillations of light. Frances C. Cogan.

Haig, Charles. **The course of rod dark adaptation as influenced by the intensity and duration of pre-adaptation to light.** Jour. Gen. Physiology, 1941, v. 24, July 20, pp. 735-751.

Adaptation tests were conducted on some subjects by employing artificial pupils, and on one subject with Argyll Robertson pupils. The findings are substantially as follows: Increase in light adaptation causes decrease in the slope of the subsequent rod dark-adaptation function and displacement of the function to the right on the time axis. Over a wide range, these changes occur to the same extent whether increase in light adaptation is produced by raising the intensity or by prolonging the exposure. Over a still wider range, dark adaptation has the same course following brief exposure to a bright light as it has following prolonged exposure to a dim light, provided the degree of light adaptation is the same in both instances. John C. Long.

Jahn, T. L., and Wulff, V. J. **Influence of a visual diurnal rhythm on flicker-response contours of Dytiscus.** Proc. Soc. Exper. Biol. and Med., 1941, v. 48, Dec., pp. 660-665.

Flicker response was determined on

the diving beetle, Dytiscus, by recording the electric potential of the eye with a cathode-ray oscillograph. The following conclusions were reached: The flicker fusion frequency-intensity relations differ for the light-adapted day-phase and night-phase eyes. This difference is marked in the high-intensity range and decreases as the threshold intensities are approached. In view of data published elsewhere the reversal of sensitivity relationships indicates that the process of light adaptation is much more pronounced in the night-phase than in the day-phase eye. The flicker-response contour and other relationships confirm the conclusion that the diurnal rhythm present in the eyes of these beetles results in two physiologic states which are so different that they may be considered functionally as separate and distinct.

John C. Long.

Jahn, T. L., and Wulff, V. J. **Retinal-pigment distribution in relation to a diurnal rhythm in the compound eye of Dytiscus.** Proc. Soc. Exper. Biol. and Med., 1941, v. 48, Dec., pp. 656-660.

The diving beetle, Dytiscus, demonstrates a persistent diurnal rhythm by the periodic migration of retinal pigment. Comparison of radial sections through the eyes of the beetles demonstrates that the distribution of retinal pigment is similar in the dark-adapted day-phase and the dark-adapted night-phase eyes. The distribution of pigment is similar in the light-adapted day-phase and light-adapted night-phase eyes, as well as in the illuminated and non-illuminated eyes of the same animal. The diurnal variations of visual function, as described in a previous article, are therefore independent of retinal-pigment migration and originate in the physiologic mechanism of the visual process. John C. Long.

Koke, M. P. **Mechanism of accommodation.** Arch. of Ophth., 1942, v. 27, May, pp. 950-968.

The theories on the mechanism of accommodation are reviewed and its present-day status is presented briefly. A method is described whereby the interior of an intact living eye may be made radiopaque by the intraocular injection of a colloidal suspension of thorium dioxide (thorotrast) and thereby pictured on roentgenogram. The roentgenologic findings were corroborated by gross inspection and microscopic studies. Roentgenograms were made of cat eyes while under the influence of cyclotonics and cycloplegics. The mechanism of accommodation was studied and compared with the probable mechanism in the human eye. Three distinct divisions of the vitreous were disclosed in the roentgenograms of adult cat eyes. In the cat eye, accommodation is brought about by compression of the posterior periphery of the lens when the tertiary vitreous is forced anteriorly and axialward by the ciliary muscle. The method of taking the roentgenograms is shown in a drawing and the findings are illustrated by photomicrographs and roentgenograms. The influence of cyclotonics and cycloplegics is shown by composite stereoscopic drawings of roentgenograms. J. Hewitt Judd.

Luckiesh, M., and Moss, F. K. **The eyelid reflex in emmetropia.** Brit. Jour. Ophth., 1942, v. 26, April, pp. 153-158.

The rate of blinking has been shown to be an unusually sensitive and reliable indicator of the ease of vision. The rate of involuntary blinking in-

creases as a visual task becomes more difficult. In order to test the theory that normal adult eyes are actually myopic under conditions of accommodative rest, the rate of blinking was measured with emmetropic subjects wearing plano lenses and —0.75-D. spherical lenses. It was thought that the addition of the minus lenses made seeing at a distance easier, since the average rate of blinking was 7.5 percent less with the myopic correction.

A reading test with additions of plus or minus 0.50 sphere showed that the lowest rate of blinking occurred with the plano lenses and that the rate was increased nearly 50 percent by the addition of either plus or minus lenses. Emmetropia is defined as "the normal state of adult nonpresbyopic eyes in which convergence makes any point within the range of habitual near-vision conjugate with the retina without exercise of relative accommodation, while more distant points may be made conjugate only by the exercise of negative relative accommodation." (2 tables, references.) Edna M. Reynolds.

Schwichtenberg, A. H. **Review of color vision.** Arch. of Ophth., 1942, v. 27, May, pp. 887-898.

A brief review of the Young-Helmholtz and Edridge-Green theories and classifications of color-vision defects is presented, and a modified technique for the Holmgren yarn test is given. A technique for the Williams lantern test is suggested, simulating conditions of military operations more closely than other tests of color vision. Results of a study made at the School of Aviation Medicine indicate quite clearly that about three fourths of the examinees who made mistakes on the Ishihara plates could not be qualified or disqualified on the results of the Holmgren yarn test used as a "confirmatory test." It was found that the Ishihara test did require a confirmatory test since 20 percent of those who made mistakes on the Ishihara test did not make significant errors on the Williams lantern test and therefore could be qualified. For all practical purposes it is reasonable to assume that such applicants will not make mistakes under ordinary circumstances. It is recommended that the Williams lantern test be used in place of the Holmgren yarn test as a confirmatory test since it is simpler, several examinees can be tested at once, and, with the technique outlined, a written record is available which can be compared with the key. The examiner's color vision does not complicate the picture. A simplified modification of the Williams lantern has been submitted to higher authority for consideration. J. Hewitt Judd.

4

OCULAR MOVEMENTS

Argañaraz, Raúl. **Functional strabismus.** La Semana Med., 1942, v. 49, April 2, pp. 629-642.

The author condemns Worth's statement that the essential cause of squint is a defect of the fusion faculty. The defect of the fusion faculty is attributed by Argañaraz to the fact that the primary deviation of the ocular axes prevents normal development of the faculty. He includes tenotomy in the general group of procedures which may be used to correct strabismus. In performing a tenotomy, he makes a vertical incision in the conjunctiva, sections the muscle and opens the capsule of Tenon, and then closes the conjunctiva horizontally instead of vertically. The

author's illustrations include photographs, before and after operation, of a 30° convergent strabismus treated by tenotomy without advancement of the other side, a case treated by double tenotomy of the internal recti and slight capsular division, tenotomy and advancement for capsulomuscular shortening according to Lagleyze, and double advancement of the external recti according to Lagleyze.

W. H. Crisp.

Davis, W. T. **Differential diagnosis of the tropias.** Amer. Jour. Ophth., 1942., v. 25, June, pp. 697-704.

Dicke, D. E. **Anomalous retinal correspondence.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 585-587.

Hagedoorn, A. **A new diagnostic motility scheme.** Amer. Jour. Ophth., 1942, v. 25, June, pp. 726-728. (One illustration.)

Holste, A. **Comparison of refraction in homozygous and heterozygous twins.** Graefe's Arch., 1940, v. 142, pt. 4, pp. 467-474.

The author examined 150 pairs of twins, in different age groups, but each pair being of the same sex. He was able to obtain details of refraction of 120 pairs, half of which were heterozygous and half homozygous. Measurements were made of the corneal diameters, corneal refractions, and total refractions. The results are shown graphically. As was to be expected, a much higher correlation of identical refractions was found in homozygous than in heterozygous twins; the inherited nature of refractive errors being thus illustrated. But there were exceptions, and the author concludes that, while similarity or dissimilarity in refraction may be contributory evidence in determining genetic identity of twins, the evidence must be weighed with that derived from other characteristics in the eye and elsewhere.

Frances C. Cogan.

Ohm, J. **Jerk nystagmus during accommodation.** Graefe's Arch., 1940, v. 142, pt. 4, pp. 401-408.

Nystagmus was studied in six cases with disturbances of the vestibular and central nervous systems. Cases 1 and 2 had multiple sclerosis principally affecting the vestibular centers. Cases 3, 4, and 5 suffered from disturbance of the peripheral vestibular system following middle-ear disease. Case 6 had spinal-cord disease of unknown origin, with signs in the central nervous system suggesting multiple sclerosis.

Spontaneous nystagmus in the light was found only in case 1. With the direction of the gaze off center it was observed in cases 1, 2, 3, and 4, and very slightly in cases 5 and 6. In the dark, nystagmus was demonstrated in cases 1, 4, 5, and 6 with the author's apparatus. (See many previous articles.) According to the author, nystagmus can be demonstrated in the dark in early cases of vestibular disturbance when it cannot be observed in the light. There was disturbance of opticokinetic nystagmus in all the cases except one which was not examined. A decrease in frequency, amplitude, and energy of the nystagmus was taken as indicative of such disturbance. Jerk nystagmus was found in all six cases during accommodation. The amplitude of the nystagmus was so small that it was necessary to use the author's apparatus to demonstrate it. Nystagmus during accommodation has not been previously recognized as evidence of vestibular and central-nervous-system disease.

To understand the relation of these various types of nystagmus (vestibular,

opticokinetic, voluntary) the author assumes that there must be a common center in the neighborhood of the vestibular nucleus.

Frances C. Cogan.

Ohm, J. **Production of opticokinetic nystagmus and of voluntary opticokinetic nystagmus.** Graefe's Arch., 1940, v. 142, pt. 4, pp. 482-494.

In the first part of this report, the author presents the technical criteria necessary for quantitative comparisons of opticokinetic nystagmus: (1) breadth of stripes, (2) rate of movement of drum, and (3) angle subtended. He also describes the production of pendular opticokinetic nystagmus.

In the second part, the author reports observations with his large drum, containing 130 white and black stripes. He found there was no simple relation, as assumed by the classic theory of Barany, between the frequency or amplitude of opticokinetic nystagmus and number of stripes. Barany's relationship only holds when two stripes are simultaneously visible and the rate of rotation is relatively slow.

Frances C. Cogan.

5

CONJUNCTIVA

Hoffa, E. L., and Smellie, E. W. **Sulfapyridine in ophthalmia neonatorum.** Brit. Med. Jour., 1942, March 7, p. 323.

A résumé of the literature is first given. Of a total of 322 cases, 273 were treated by sulfapyridine by mouth, most of the remaining 49 constituting an earlier group treated by local medication. The incidence of gonococcal infection was 27.3 percent, there being 89 gonococci-positive cases and 233 gonococci-negative. Thick purulent discharge was present in 93 cases; 36 of these were gonorrheal and 57 nongonorrheal. Sulfapyridine was used irrespective of the causal organism, and clinically there appeared to be no obvious specially selective action on gonococci. Sixty-two percent of the cases were cured clinically within eight days by such administration, against 15 percent by local methods. The fact that as many rapid recoveries under sulfapyridine are observed in nongonorrheal as in gonorrheal cases is noteworthy. The differential incidence of organisms other than gonococci is of interest. Pus cells only, without any organisms in smear or culture occurred 53 times and the remaining 180 cases showed the following; staphylococcus, 111; other cocci, 20; and bacilli, 49. (Peculiarly, the article makes no mention of inclusion-body blennorrhea.)

The management of cases is as follows. On admission a swab of the pus is taken, both for a smear preparation and for culture. The eyes are then irrigated with a bland lotion, such as 0.5 percent saline solution. Atropine sulphate 1 percent and drops of medicinal paraffin are instilled as routine measures in all cases, and half a tablet of sulfapyridine (0.25 gm.) crushed into powder is given by mouth in a teaspoonful of water or milk. Sulfapyridine administration is continued in doses of 0.125 gm. every four hours, day and night, until 24 hours after a clinical cure is obtained. Usually this means treatment for three to five days, with a total dosage of about 2.5 to 4 gm. Local treatment consists of three-hourly irrigation with saline solution during the first day in cases with profuse discharge; as a rule there is no need for further irrigation on the subsequent days. After irrigation medicinal paraffin is instilled as a precaution against sticking together of the lids. Atropine

is instilled three times daily in cases with corneal haze or ulceration. (20 references.) Ralph W. Danielson.

Klauder, J. V., and Cowan, A. **Ocular pemphigus and its relation to pemphigus of the skin and mucous membranes.** Amer. Jour. Ophth., 1942, v. 25, June, pp. 643-662; also Trans. Amer. Ophth. Soc., 1941, v. 39, p. 277. (10 illustrations, bibliography.)

Kluever, H. C., Coughlan, C. H., and Martin, L. M. **Hypersensitiveness of the conjunctiva and cornea.** Jour. Iowa State Med. Soc., 1941, v. 31, Dec., p. 572.

A case report with discussion of a violent hypersensitiveness of the conjunctiva and cornea. Offending substances included atropine, silver nitrate, mercury compounds, typhoid vaccine, staphylococcus toxin, and many foods.
Theodore M. Shapira.

Páez Allende, Francisco. **Rare nevus of the conjunctiva.** Anales Argentinos de Oft., 1941, v. 2, June, pp. 90-98.

The nevus, which bordered on the upper limbus, measured 12 mm. horizontally and 6 mm. vertically. Over a length of 4 or 5 mm. the cornea was invaded by the nevus to the extent of 1 mm. The nevus contained a cystic formation about 2 mm. in diameter.

The patient had another nevus of the face, near the left angle of the mouth. The growth on the eyeball was apparently congenital, and the patient was advised to report in case the growth became larger. (5 illustrations.)
W. H. Crisp.

Wong, R. T. **Chemotherapy in the treatment of gonorrheal ophthalmia.** Arch. of Ophth., 1942, v. 27, April, pp. 670-687.

The author gives a historical review of the literature and discusses the mode of action, toxic reactions, and major complications of sulfanilamide therapy. He summarizes the toxic manifestations of sulfapyridine and sulfathiazole and the ocular manifestations of toxicity of sulfanilamide and its derivatives. Dosage and adjuvant treatment are discussed. Forty cases of gonorrheal ophthalmia were treated; 20 with sulfanilamide, 10 with sulfapyridine, and 10 with sulfathiazole. Criteria of cure were arrest of discharge and subsidence of the inflammation, obtaining three consecutive daily negative smears of the conjunctival secretion and, in the sulfathiazole group, obtaining one negative epithelial smear beside the negative smears of the secretion. Of the cases treated with sulfanilamide, 18 cases were cured within eight days. Sulfapyridine was found to be more effective than sulfanilamide, but sulfathiazole seemed to be superior to both, since in seven cases, gonococci disappeared from the secretion in from 12 to 24 hours after treatment was instituted. In all the cases, the swelling and inflammation subsided and the discharge lessened within 12 hours. The average time required for cure was 3.4 days and the maximum time was four days. Sulfathiazole was not only more efficient therapeutically, but it showed a lower incidence of toxic reaction.
J. Hewitt Judd.

6
CORNEA AND SCLERA

Cogan, D. G., and Kinsey, V. E. **The cornea. 2. Transfer of water and sodium chloride by hydrostatic pressure through the excised cornea.** Arch. of Ophth., 1942, v. 27, April, pp. 696-704.

The authors describe the experiments by which excised corneas, prepared in a manner similar to that employed for

their experiments with osmosis and diffusion, were used for studies to compare the amount of fluid transferred by a given hydrostatic pressure through cornea before and after removal of its constituent membranes; to compare the amount of fluid transferred through the intact cornea, employing different hydrostatic pressures; and to determine the osmotic equivalent of a given hydrostatic pressure. They found that the excised cornea is impermeable to 1-percent sodium-chloride solution in the out-in direction with hydrostatic pressures up to at least 400 mm. of mercury; this impermeability depends on the integrity of the epithelium. The excised cornea is permeable to 1-percent sodium-choride solution in the in-out direction under various hydrostatic pressures; this permeability undoubtedly depends on the epithelial damage resulting from the hydrostatic pressure. The amount of fluid when transferred through the cornea is directly proportional to the hydrostatic pressures employed. The corneal epithelium possesses a high degree of semipermeability with respect to sodium-chloride solutions for a biologic membrane. The authors found that, despite the histologic similarity of the epithelium of conjunctiva and of cornea, the excised conjunctiva did not function appreciably as a semipermeable membrane with sodium chloride, and they point out that this differential permeability of cornea and conjunctiva has obvious clinical implications with regard to the absorption of therapeutic agents.

J. Hewitt Judd.

Cosgrove, K. W., and Day, P. L. **The use of riboflavin in the treatment of corneal diseases.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 544-551. (2 tables, bibliography.)

Kluever, H. C., Coughlan, C. H., and Martin, L. M. **Hypersensitiveness of the conjunctiva and cornea.** Jour. Iowa State Med. Soc., 1941, v. 31, Dec., p. 572. (See Section 5, Conjunctiva.)

Sherman, A. R. **Crystalline dystrophy of the cornea.** Arch. of Ophth., 1942, v. 27, April, pp. 692-695.

In a man aged 45 years slitlamp examination of both eyes revealed the presence of countless minute varicolored crystals in the anterior portion of the cornea, closely packed in and beneath Bowman's membrane, and more sparsely distributed through the remaining anterior third of the cornea. They extended from limbus to limbus in all directions, although slightly less in the periphery than elsewhere, and in the light of the slitlamp glistened brilliantly. With the ocular micrometer in the microscope they measured 0.02 to 0.05 mm. The condition has remained unchanged for five years. There were no opacities, inflammatory signs, or visual disturbances. (One drawing.)

J. Hewitt Judd.

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Eggers, Harry. **Experiences with iridencleisis.** Arch. of Ophth., 1942, v. 27, April, pp. 665-669.

Eggers reviews Holth's description of his operative technique and points out that in order for the operation to be entirely successful, it is necessary to have a properly prepared flap. In the author's technique the initial incision is made about 7 mm. above the limbus in a slightly curved direction with concavity toward the limbus. This is made directly down to the sclera, and is extended on either side until it reaches

a little beyond the projection of the medial and temporal edges of the cornea. A trapezoidal area is undermined down to within 0.5 mm. of the limbus in the midline. The author feels that avoidance of continuing the flap into the cornea produces a thicker and therefore safer flap. A keratome incision is made 2 mm. behind the limbus. A small sclerectomy is done on the corneal lip, the iris is pulled through the scleral incision, pigmented surface up, and two radial incisions about 2.5 to 3 mm. apart are made through the iris from the pupillary margin to the base. The iris tongue is smoothed out, the conjunctival flap replaced and sutured. J. Hewitt Judd.

Leathart, P. W. **The tonic pupil syndrome.** Brit. Jour. Ophth., 1942, v. 26, Feb., p. 60.

This syndrome has been previously described under the titles "nonluetic Argyll Robertson pupil" and "partial iridoplegia." No lesion has been suggested which is capable of producing the physical signs described. The syndrome is recognized by three physical signs, each of which points to some interference with the nervous mechanism of the intrinsic muscles of the eyes. (1) The pupillary reaction to light is absent or sluggish. (2) The affected pupil is larger than its fellow but will dilate further in the dark, and upon use of atropine. (3) The pupillary reaction to convergence is slower than normal in spite of the fact that near vision is unaffected. The rest of the eye is normal in all respects. There is no external muscular paresis, no ptosis or nystagmus, and no enophthalmos or exophthalmos. In a certain number of cases, the knee jerks and ankle jerks are absent or reduced.

The author postulates a destructive lesion in the ciliary ganglion, giving rise to false conduction between preganglionic and postganglionic parasympathetic fibers. "Ciliary ganglion iridoplegia" is suggested as a more correct name of the phenomenon.

Edna M. Reynolds.

Verhoeff, F. H. **Retinitis pigmentosa with widespread gliosis—so-called choroideremia.** Arch. of Ophth., 1942, v. 27, April, pp. 688-691. (See Section 10, Retina and vitreous.)

8

GLAUCOMA AND OCULAR TENSION

Barkan, Otto. **Operation for congenital glaucoma.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 552-567. (10 illustrations, references.) Also Trans. Pacific Coast Oto-Ophth. Soc., 1941, 29th mtg.

Schoenberg, M. J. **The problem of preventing partial or total loss of vision in glaucoma patients of eye clinics.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 521-524; also Trans. Amer. Acad. Ophth. and Otolaryng., 1941, 46th mtg., Nov.-Dec., p. 61.

Schoenberg, M. J. **Survey of records of glaucoma in ophthalmic clinics.** Arch. of Ophth., 1942, v. 27, April, pp. 716-727; also Trans. Amer. Ophth. Soc., 1941, v. 39, p. 161.

A survey form with instructions for use, prepared by the committee on glaucoma of the National Society for the Prevention of Blindness, is presented together with the results of surveys made of case records of glaucoma in four ophthalmic clinics. Because of many deficiencies found in the records, it was impossible to get complete information regarding history, methods of examination, clinical findings and

results. The author recommends that, where the number of glaucoma patients warrants it, a special glaucoma clinic be organized, with an experienced ophthalmologist in full charge. The unit should comprise clerical, ophthalmologic, technical, nursing, social-service, consultant, and laboratory components. A printed record form such as that presented should be adopted, and an efficient system for registering the patients and filing and cross-indexing the records should be instituted. The attending staff should be trained for early recognition of glaucoma. A survey of all phases of glaucoma work in the institution should be made annually and the results compared with those of previous years. J. Hewitt Judd.

Sugar, H. S. **The practical applications of gonioscopy to glaucoma surgery.** Amer. Jour. Ophth., 1942, v. 25, June, pp. 663-671. (References.)

9

CRYSTALLINE LENS

Adams, P. H. **Doyne's discoid cataract.** Brit. Jour. Ophth., 1942, v. 26, April, p. 152.

An ophthalmoscopic and a slitlamp drawing of a case of discoid cataract are presented. Edna M. Reynolds.

Bertotto, E. V. **Visibility of the retrolenticular space after operation for retinal detachment.** Anales Argentinos de Oft., 1941, v. 2, June, pp. 85-89. (See Section 10, Retina and vitreous.)

Damel, C. S., and Teruggi, A. **Subconjunctival luxation of the lens.** Arch. de Oft. de Buenos Aires, 1941, v. 16, July, p. 400. (See Section 16, Injuries.)

Klein, M. **Investigation concerning the lens capsule. Its importance in the technique of intracapsular cataract extraction.** Brit. Jour. Ophth., 1942, v. 26, March, pp. 118-126.

The anatomic and physiologic aspects of intracapsular extraction are discussed. The zonular lamella is a very thin and elastic membrane covering the peripheral parts of the anterior lens capsule, which can easily be separated from it. The zonular fibers are attached to the zonular lamella and not to the lens capsule proper.

The thickness of the lens capsule is not uniform. The posterior capsule is thinner than the anterior. In both the anterior and posterior capsules the central area is thinnest. The maximum thickness of the capsule lies between the equator and the center concentrically. The thickness of normal capsules varies with age.

The zonular fibers arise from the whole area of the ciliary body. The shorter fibers coming from its anterior parts pass straight to the lens through the posterior chamber. The longer ones which come from the back of the ciliary body are applied firmly to the vitreous or are embedded in it. On the lens these are attached to the zonular lamella. With advancing years, the zonular fibers become fragile and the lens capsule gets thicker.

The author outlines a method of investigation of the tensile strength of the lens capsule. He finds that the cataractous lens capsule can resist a pressure of from 150 to 200 mm. Hg.

In separating the lens from the zonule in intracapsular operation, the author recommends traction on the lens capsule and simultaneous pressure on the sclera at the limbus with a squint hook in the direction of the traction. This procedure is repeated in different directions so as to separate the zonule from the lens, section by section. The author suggests opening the forceps

only 3 or 4 mm. and using a capsule forceps which will grip without tearing or slipping off. The suction cup is recommended for tumescent or hypermature cataracts. (2 figures, references.) Edna M. Reynolds

Klein, M. **Operation for cataract.** Brit. Jour. Ophth., 1942, v. 26, March, pp. 93-117.

A review of 650 cataract operations performed in the Ophthalmic Department of the Jewish Hospital in Budapest between 1933 and 1938 is given. The preoperative treatment, method of anesthesia and operative technique are discussed.

It was found that the cases in which the corneoscleral-stitch method of wound closure was used required the shortest postoperative hospital treatment. Visual acuity was better after conjunctival flap and corneoscleral-stitch operations than after those in which Kuhnt's apron was employed. Vitreous complication was slightly more frequent with intracapsular extraction. Vitreous complication during extraction was found to have practically no effect upon the eye. It is the author's opinion that the importance of vitreous loss has been exaggerated. Iris prolapse occurred twice as often after extracapsular operation as after intracapsular. The type of iridectomy appeared to have no bearing on the occurrence of iris prolapse. The outcome of the cases with iris prolapse was relatively good.

Increase of intraocular pressure occurred far more frequently after extracapsular than after intracapsular extraction. Postoperative infections were also much more common after extracapsular extraction. The value of reinforced wound closure and the superiority of the intracapsular method of extraction are emphasized. (2 figures, 4 tables, references.)

Edna M. Reynolds.

Verhoeff, F. H. **A simple and safe method for removing a cataract dislocated into fluid vitreous.** Amer. Jour. Ophth., 1942, v. 25, June, p. 725.

Von der Heydt, Robert. **Visual prognosis for the aging lens.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 576-578.

10

RETINA AND VITREOUS

Ascott, J. R. **A case of conjunctival implantation cyst following operation for detachment of retina.** Brit. Jour. Ophth., 1942, v. 26, March, pp. 126-128.

The cyst measured 5/16 in. by 3/16 in. and projected sufficiently to cause a definite fulness in the lower lid. The deepest part of the cyst was found at operation to extend to the insertion of the inferior rectus, where an unabsorbable suture was removed. It was believed however, that the cyst was unrelated to the suture and was the result of implantation of conjunctival epithelium which had occurred while a suture was being drawn through the conjunctiva from without inward. (One drawing.) Edna M. Reynolds.

Balado, M., and Fortin, E. P. **Topography of the fibers in the optic nerve in relation to retinal regions.** Anales Argentinos de Oft., 1941, v. 11, July-Aug.-Sept., p. 135. (See Section 11, Optic nerve and toxic amblyopias.)

Berens, Conrad. **Scleral hooks.** Amer. Jour. Ophth., 1942, v. 25, May, p. 588. (One illustration.)

Bertotto, E. V. **Visibility of the retrolenticular space after operation for retinal detachment.** Anales Argentinos de Oft., 1941, v. 2, June, pp. 85-89.

Two disinsertions in the same eye, one extending from the four to the eight-o'clock position and the other from the nine to the twelve-o'clock position, had been operated upon by perforating diathermy. Two months after operation the eye had vision equal to unity, although there were still some areas of slight retinal elevation. A further operation was performed nearly four months after the first. Three weeks later there was an apparent opacity in the anterior vitreous, and four weeks afterward the existence of the retrolenticular space was made out. About a year after the second operation, there were numerous pigmented deposits on the posterior surface of the crystalline lens. Between this surface and the anterior surface of the posterior wall of the retrolenticular space there were connective tissue fibers in which pigment was deposited. The posterior surface of the hyaloid was thickened. Visual acuity had fallen to 1/4. Two months later the vision had recovered to 2/3. W. H. Crisp.

Crawford, J. W. **Familial degeneration of the macula lutea.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 525-543; also Trans. Amer. Ophth. Soc., 1941, v. 39, p. 344. (3 figures, case reports, bibliography.)

Damel, C. S., Gallino, J. A., and Travi, O. A. **Disciform degeneration of the macula.** Arch. de Oft. de Buenos Aires, 1941, v. 16, July, p. 376.

The authors report three cases of disciform degeneration of the macula of the senile type, one of which showed a concomitant retinitis circinata. The literature is reviewed in detail. (Fundus photographs, bibliography.)

Plinio Montalván.

Hagen, P. S. **The retinal manifestations of subacute bacterial endocarditis.** Minnesota Med., 1941, v. 24, April, p. 237.

A study of 37 cases reveals that nerve-head lesions occurred in 35 percent. Further, petechiae are more consistently found in cases with disc pathology. Marked bilateral papilledema may be present in vegetative endocarditis with elevation in spinal-fluid pressure.

Theodore M. Shapira.

Linksz, Arthur. **Improved model of the Kukán ophthalmodynamometer.** Amer. Jour. Ophth., 1942, v. 25, June, pp. 705-713. (10 figures, references.)

McKee, S. H., Wilson, D. R., Fowler, A. F., and Wilen, B. **Lipemia retinalis.** Amer. Jour. Ophth., 1942, v. 25, May, pp. 513-521. (Illustrations, tables, references.)

Rubino, A. **A peculiar bilateral and symmetric anomaly of the pigment layer of the retina.** Boll. d'Ocul., 1940, v. 19, April, pp. 318-322.

A girl of 15 years showed a starfish-like figure of the fundus, having the optic disc at its center, and with four arms, three of which were short and coniform while the upper one ended at the margin of the retina. The color was reddish yellow and three of the arms contained islets of pigments of varying size. The retinal vessels passed over the figure in their regular course. Both eyes were affected alike. The writer discusses the embryogenesis of the anomaly. He thinks it due to atrophy of the pigment layer with anomalous distribution of pigment, and he calls it "dysgenesis circumpapillaris strati pigmenti retinae." (One colored figure.) M. Lombardo.

Tooke, F. T., and Nicholls, J. V. V. **On the character and coincidence of retinal hemorrhages occurring in diabetes.** Canadian Med. Assoc. Jour., 1942, v. 46, Jan., p. 35.

Ophthalmoscopic studies were made on one hundred patients, 23 of whom had retinal hemorrhage. A table is presented showing age, sex, blood pressure, and blood-chemistry and urinary findings, and comparing one hundred diabetic with one hundred primary hypertensive cases with particular reference to retinal hemorrhage. Thirty-one cases had definitely high blood pressure but no hemorrhage, while nine cases with hemorrhage displayed normal blood pressure. This suggested some element other than cardiovascular hypertension as responsible for the terminal changes.

The discussion of retinal disease in diabetics seems to place the authors among those who feel that such retinitis is characteristic and distinguishable from arteriosclerotic or renal retinopathies. F. M. Crage.

Verhoeff, F. H. **Retinitis pigmentosa with widespread gliosis—so-called choroideremia.** Arch. of Ophth., 1942, v. 27, April, pp. 688-691.

True choroideremia, which is a congenital absence of the choroid except behind the macula, is analogous to coloboma of the macula and probably can occur. However it has never been observed microscopically and it is not certain that the anomaly has ever actually occurred. The author points out that many cases of supposed choroideremia are really cases of retinitis pigmentosa in which the choroid is hidden by widespread gliosis of the degenerated neuroepithelial layer of the retina. In rare cases the gliosis involves the whole extent of the retina with the exception of the macula. The author suggests the term pseudo-choroideremia for such cases. He discusses briefly his findings in a case already reported, and examined microscopically. He also mentions eight cases in two families (seen by Waite and Gundersen), in one of which cases the condition was first thought to be choroideremia, but in which the white patches in the fundus were found not to be exposed sclera, but deep-seated retinal opacities probably caused by retinal gliosis. J. Hewitt Judd.

Weskamp, Carlos. **Chorioretinitis serosa macularis.** Anales Argentinos de Oft., 1941, v. 11, July-Aug.-Sept., p. 143. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

11
OPTIC NERVE AND TOXIC AMBLYOPIAS

Balado, M., and Fortin, E. P. **Topography of the fibers in the optic nerve in relation to retinal regions.** Anales Argentinos de Oft., 1941, v. 11, July-Aug.-Sept., p. 135.

The authors discuss the work already published on this subject and offer a scheme which demonstrates their own views. The fibers arising from close to the disc lie in the periphery of the nerve, those more central lie next to these, and the fibers arising peripherally lie in the center of the nerve. As in all investigations of this subject, many points remain to be explained, and can only be solved by a combination of anatomical studies, visual-field exploration, and clinical observation. Eugene M. Blake.

Bedell, A. J. **Papilledema without increased intracranial pressure.** Amer. Jour. Ophth., 1942, v. 25, June, pp. 685-696; also Trans. Amer. Ophth. Soc.,

1941, v. 39, p. 242. (10 illustrations, bibliography.)

Dixon, G. J. **The significance of a lesion of the optic nerve.** Brit. Jour. Ophth., 1942, v. 26, Feb., p. 64.

The survey covers 27 optic-nerve lesions seen at the Royal Westminster Ophthalmic Hospital in 1931. Twenty-four patients had a complete clinical examination. Of this group, six patients were found to have indisputable evidence of disseminated sclerosis, five had tabes dorsalis, two had cerebral tumor, and one had received an injury. In two cases, after ten years of observation it was decided that the optic-nerve lesion was due to arteriolar sclerosis. One patient developed disseminated sclerosis two years after onset of the retrobulbar neuritis. The cause of the nerve lesion was unknown in seven patients. Two cases recovered spontaneously.

Edna M. Reynolds.

Ford, Rosa. **The diagnostic significance of retrobulbar neuritis.** Brit. Jour. Ophth., 1942, v. 26, March, pp. 128-130.

A case in which retrobulbar neuritis was the precursor of iritis (five-weeks duration) and of rheumatoid arthritis (five-years duration) is reported. On the supposition that a septic focus might exist in the sinuses in spite of the absence of nasal symptoms, the sinuses were drained. The iritis promptly cleared and the arthritis was brought to a standstill, with freedom from pain. One sign of closed sinusitis is contraction of the visual fields. Thus the ophthalmologist has a role in prevention as well as cure of later disease of such origin. (References.)

Edna M. Reynolds.

Ford, Rosa. **The etiology of an obscure case of retrobulbar neuritis.** Brit. Jour. Ophth., 1942, v. 26, Feb., p. 56.

A case of retrobulbar neuritis of obscure origin was diagnosed by the ophthalmologist after seven months as being due to a concealed sinusitis. The diagnosis was based on two considerations: (1) A bad cold accompanied by double ptosis, marked photophobia, and severe headache preceded the visual loss. (2) In each eye the field showed marked contraction, approaching the fixation point above. Study by a rhinologist had been delayed for seven months, because neither sign nor symptom of sinusitis was present. A rhinologist then found pus in the sphenoidal sinus, and produced immediate improvement in vision. But the patient failed to make a complete recovery until five years later, when the apparently normal antra were also opened and were found to be full of pus. A negative rhinologic examination does not end the ophthalmologist's responsibility. Edna M. Reynolds.

Giqueax, R. E. **Post-hemorrhagic amaurosis.** Anales Argentinos de Oft., 1941, v. 2, July-Aug.-Sept., p. 155.

The case cited by the author was that of a 56-year-old man who presented himself with dilated pupils, hyperemic nerve heads with blurred borders, and blindness of both eyes. A provisional diagnosis of optic neuritis was made but a complete physical examination revealed no changes except a recent severe intestinal hemorrhage. In spite of transfusions, vitamins, and deep orbital injections of atropine for 15 days the nerves went on to complete atrophy. A very good review of this rather unusual complication of a frequent accident—severe loss of blood—is appended. Eugene M. Blake.

Noran, H. H., and Polan, C. G. **Devic's disease (ophthalmoneuromyelitis).**

Arch. of Ophth., 1942, v. 27, April, pp. 707-715.

The clinical picture and pathologic changes found in this condition are reviewed and the case of a woman aged 48 years is reported. The case is rather unusual, in that the ascending myelitis involved the cervical portion of the cord with respiratory difficulty, and resulted in terminal respiratory and cardiac failure. Pathologic findings at autopsy revealed a lack of glial repair, absence of plaque formation, and absence of changes in the brain stem, cerebellum, and cerebrum, thus making a diagnosis of acute multiple sclerosis unlikely. Acute disseminated encephalomyelitis was ruled out by the absence of widespread perivascular demyelinization. The limitation of the involvement to the optic nerves and the spinal cord plus the diffuse nature of the lesions with an absence of glial repair seems to favor the conception that acute neuromyelitis optica may be a disease entity rather than an acute form of disseminated sclerosis or encephalomyelitis. (4 photomicrographs.)

J. Hewitt Judd.

NEWS ITEMS

Edited by Dr. Ralph H. Miller
803 Carew Tower, Cincinnati

News items should reach the editor by the twelfth of the month.

Deaths

Dr. Lambert H. Weinheimer, Syracuse, New York, died April 7, 1942, aged 54 years.

Dr. Leo Wolfenstein, Cleveland, Ohio, died April 3, 1942, aged 68 years.

Dr. Abraham David Eisenberg, Brooklyn, New York, died April 3, 1942, aged 60 years.

Dr. Joseph Wilbur Shelar, Mount Pleasant, Pennsylvania, died April 18, 1942, aged 82 years.

Dr. Ernest B. Baker, Edcouch, Texas, died in April, 1942, aged 70 years.

Dr. Keith Maurice Morris, Saginaw, Michigan, died in April, 1942, aged 59 years.

Dr. John B. Alcorn, Columbus, Ohio, died June 16, 1942, aged 69 years.

Dr. Arthur Donald Wilson, Mission, Texas, died April 17, 1942, aged 38 years.

Dr. Charles Hunter Robinson, Cullom, Illinois, died April 14, 1942, aged 66 years.

Dr. James Monroe Sleicher, Chehalis, Washington, died April 6, 1942, aged 88 years.

Dr. James William Wilkins, Mount Olive, North Carolina, died April 3, 1942, aged 55 years.

Dr. Albert Fowler Rodrick, Swampscott, Massachusetts, died April 30, 1942, aged 69 years.

Dr. Frederick Eugene Vaughan, Mount Kisco, New York, died April 14, 1942, aged 57 years.

Dr. Jason Newell Robertson, Wolcott, New York, died April 7, 1942, aged 88 years.

Miscellaneous

The following course of postgraduate lectures in ophthalmology is to be held in the Tennent Institute of Ophthalmology, Western Infirmary, Glasgow, Scotland: September 9th, Professor Riddell—"Certain aspects of retinal function"; September 16th, Dr. Bacsich—"Comparative anatomy of the retina"; September 23d, Professor Loewenstein—"Lipoid degeneration of the retina"; September 30th, Professor Ballantyne—"Venous system of the retina"; October 1st, Dr. Eugene Wolff—"Normal histology of the retina"; and October 2d, Dr. Eugene Wolff—"Pathologic histology of the retina."

A station for checking the accuracy of Schiötz tonometers is temporarily located at the offices of the National Society for the Prevention of Blindness, Room 1300, 1790 Broadway, New York City, under the direction of Dr. Adolph Posner. Inquiries regarding this service will be welcomed.

Societies

At an emergency meeting of the officers and council of the American Academy of Ophthalmology and Otolaryngology, Dr. William L. Benedict was appointed Executive Secretary of the Academy to fill the vacancy created by the death of Dr. William P. Wherry.

The Eastern Pennsylvania Association of

Eye, Ear, Nose, and Throat Physicians held its first annual meeting at the Schuykill Country Club on April 29th. The following officers were elected: Dr. James E. Landis, Reading, president; Dr. Charles L. Fackler, York, vice-president; and Dr. Sterling F. Mengel, Pottsville, secretary-treasurer. The program consisted of the following speakers: Drs. Benjamin F. Souders, Thomas R. Gagion, Horace J. Williams, Douglas MacFarlan, Algernon B. Reese, and Samuel R. Kaufman.

Personals

Dr. Frank H. Rodin received a grant of $20,000.00 from the Columbia Foundation to conduct Research in Ophthalmology. This work will be conducted at the Mount Zion Hospital Research Laboratories in San Francisco under the direction of Dr. Frank H. Rodin and Dr. Charles Weiss.

Trustees of the American Medical Association gave a cocktail party in the Traymore Hotel, on June 11th, in honor of the Latin Americans and their families. It was very well attended, and new cordial friendships were made not only between representatives of the different American countries and American physicians, but also between delegates themselves.

Dr. William B. Clark, New Orleans, presented a paper on "Anterior-segment eye diseases" at the annual banquet of the Emory Medical Alumni Association, June 4th.

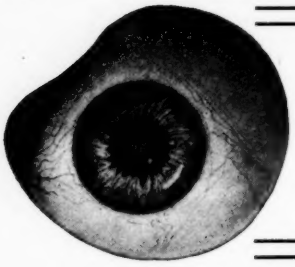
The West Virginia State Medical Association held its seventy-fifth annual meeting in Huntington, July 13th to 15th. Among the guest speakers was Dr. Ralph Irving Lloyd, who discussed "The proptosed eye as a diagnostic problem."

At the eighty-ninth annual meeting of the Minnesota State Medical Association, Dr. Ferdinand H. Haessler, Milwaukee, spoke on "Ocular tuberculosis."

Among the speakers at the annual meeting of the New Mexico Medical Society was Dr. Albert W. Egenhofer, Sante Fe, who lectured on "Eye conditions of interest to the general practitioner."

Dr. John Y. Battenfield, formerly of Pryor, Oklahoma, has been recently appointed Medical Associate of the National Society for the Prevention of Blindness.

Among the recipients of the awards made by the collegiate division of the University of Chicago in special recognition of achievements in their special fields was Dr. Edward V. L. Brown, professor of ophthalmology, University of Illinois College of Medicine.



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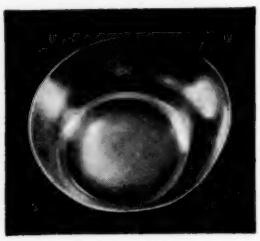

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